General
Diagnosis and Therapy
of Skin Diseases

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AN INTRODUCTION TO DERMATOLOGY
FOR STUDENTS AND PHYSICIANS

By

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WITH 375 ILLUSTRATIONS



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Preface

IN THE Preface to the German edition of his book (1952) Hermann Werner Siemens states that the only excuse for a new textbook on dermatology can be a new approach to teaching dermatology He has in mind concentration on the *fundamentals of dermatology* by thorough explanation and particularly by illustrations in close up photography Of course most textbooks devote a certain amount of space to the fundamentals namely the definition of the lesions and also the principles of therapy but these efforts have usually the character of a short introduction hurrying to the main part of the texts—the special pathology and therapy of the skin diseases There it is hoped the beginner will also find the illustrations which are to clarify the definitions in words. Of course it is most desirable to have these illustrations right next to the discussion of the lesions eruptions, and therapy and the photography should be taken with the purpose in mind of illustrating a written definition. Siemens has, in more than twenty five years of teaching at the University Skin Clinic at Leiden, directed his photographer Mr J J van der Walle to take pictures just as he found necessary for his way of teaching the elements of dermatology On the basis of a very large number of such photographs, the book has been written independently of existing texts.

In the chapters on therapy Siemens tries to inculcate in the student the fundamental rules of all therapy Use for healing what experience has shown to be effective but only that. Do not choose medications for reasons of tradition or pharmacological expectations that the method *should* be good but because you know it is good Carefully weigh effect against toxicity Throw out ruthlessly what is honored only by time and not by merit Be suspicious of complicated prescriptions, no matter how famous. Learn how to master a small, though sufficient armamentarium and stick to it, instead of becoming a therapeutic butterfly that sips from one blossom today and tomorrow from another never really familiar with anything The chapter on the application of therapeutic agents is based entirely on Siemens own clinical *experiments. He is a man who is perfectly willing to spend much of his time and his great energy* in the unglamorous work of investigating evaluating and often debunking famous recipes which have been handed down to us. He teaches that the student should not memorize ready-made *prescriptions* but, instead, learn the strengths of the agents so that he can prescribe them as the case requires. *Of course* not too much of our so-called heritage survives *this* strict treatment but what passes is better founded, *simpler and often* better to work with than the eighty combinations of old. The therapeutic part of the book is written *in a*

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spirit of scientific curiosity which changes everyday treatment from a monotonous routine to an interesting experiment. But Siemens, though sometimes tending to lean over backward in order to demonstrate his point to the beginner, is also a man of practical experience. Even the most experienced dermatologist will pick up sound advice on many pages, and he will enjoy the graphic descriptions and pictures. The best point, however, which the medical student can learn from the book is the spirit of intellectual discipline and honesty in which it is written.

Siemens, who comes from a famous German family of engineers, scientists, inventors and industrialists, received his dermatological training at the clinics of Joseph Jadassohn in Breslau and Leo von Zumbusch in Munich, where he also taught. In 1929 he was offered the chair of dermatology at the famous University of Leiden, Holland, where he trained many Dutch and other dermatologists. During the occupation of the Netherlands by the Nazis he courageously defended the academic freedom of his university. As could be expected, the invaders soon dismissed him from his position. For his opposition the Germans took him twice as a hostage and imprisoned him in a concentration camp. After the defeat of Hitler and the liberation of the Netherlands, the grateful Dutch government immediately reinstated him, and his first lecture inspired a moving demonstration of respect for the German who, even under threat of death, had placed his convictions and human principles above everything else.

Siemens has written a very large number of articles, many of them the result of thorough scientific investigation. Besides dermatology, Siemens has from his student days been fascinated by the field of human genetics. Frequently he applied his great knowledge of human genetics to dermatology. In 1923 the method of studying identical twins to confirm or rule out hereditary factors in disease was introduced by him and often applied to dermatoses. In 1924 he wrote a monograph on the method of "Twin Pathology"[1] and much of his genetic work, as far as it has a bearing on skin diseases, is presented in Jadassohn's monumental Handbook,[2] for which he wrote the chapter on heredity in dermatology.

My translation was greatly helped by Drs. Harold L. Miller, Joel F. Taxman and Roger Laubenheimer, all dermatologists of Milwaukee, who read the manuscript. Dr. Allan L. Lorincz, assistant professor of dermatology at the University of Chicago, also read the manuscript and suggested many improvements. Mr. Walter Hahn, a registered pharmacist of Milwaukee, checked the dosages and helped me to adapt some European prescriptions to American conditions. My secretary, Mrs. Gloria Kurtz, typed the manuscript. I am most grateful to every one of them.

KURT WIENER

1. *Die Zwillingspathologie, ihre Bedeutung, ihre Methodik, ihre bisherigen Ergebnisse* (Berlin: J. Springer, 1924).

2. Joseph Jadassohn, *Handbuch der Haut- und Geschlechtskrankheiten*, Vol. III (Berlin: J. Springer, 1929).

The *cutis* consists of a network of *collagenous* (meaning "glue-generating") and spirally twisted *elastic fibers* This composition from two types of fibrous elements makes the skin tough as well as expansible so that structurally it can be likened to an elastic suspender In the meshes of the fibrous elements are relatively few oblong *connective tissue cells* and in pigmented skin areas also rather large more or less star shaped cells which contain coarse granules of pigment. This pigment, however was not formed by these cells but was picked up from the basal layer of the epidermis and transported into the cutis. Thus they are merely *carriers* of pigment, called *melanophores* or *chromatophores*

The connective tissue of the cutis projects finger like processes called *papillae* into the epidermis above it Since the epidermal rete fills the space between these digitations, a system of *rete ridges* develops. Therefore in vertical cross-section, the border between epidermis and cutis appears to be wavy One should however keep in mind that only the upward-directed curves represent actual individual projections, while the depressions between them form a continuous network of ridges. (Because of the appearance in cross-section, the erroneous term *rete pegs* is often used rather than the correct term *rete ridge*) All papillae—i.e. the cutis projections—as a whole form the important uppermost part of the cutis, called the *papillary body* or the *stratum papillare* The zone beneath the papillae is termed *stratum reticulare* The histopathologist frequently needs a special word for the uppermost part of the stratum reticulare which adjoins the papillae and forms their common base. The term *stratum subpapillare* is used for this structure.

Like the epidermis, the cutis is also of widely varying *thickness* on different parts of the body surface Its tissue structure may be *loose* or *dense* to give widely varying degrees of firmness or toughness to the skin. The cutis is particularly loose on the eyelids, the dorsa of the hands, and the genitalia For this reason, these sites are liable to form excessive and even giant accumulations of fluid (edemas) In contrast to the epidermis, the cutis is everywhere well supplied with blood and lymphatic vessels.

The *blood vessels* of the cutis form a superficial (subpapillary) vascular network beneath the papillae and a deep (deep-reticular) one at the border between cutis and subcutis Both vascular networks run parallel to the surface of the skin and are connected by anastomoses, which also supply the appendages of the skin (hair follicles, sebaceous and sweat glands) The upper network sends capillary loops vertically into the papillae

In addition to the blood vessels, the cutis contains a lymphatic system In the epidermis, lymph circulates between the rete cells and fills the *intercellular spaces* Lymph also collects in the border between epidermis and cutis. In the cutis and subcutis there occur actual *lymphatic capillaries and vessels* which frequently accompany the veins.

The skin is supplied by the autonomic, as well as the cerebrospinal *nervous*

melanocytes (dendritic melanoblasts or Ehrmann s cells) which are believed to be the cells in which melanogenesis primarily occurs. The dendritic melanocytes extend upward into the entire layer of the stratum spinosum

In the *stratum spinosum* the cells have become polyhedral Protoplasmic bridges appear to cross the intercellular spaces from cell to cell giving at first glance the impression of prickles or spines. For this reason the cells of this layer have been called *prickle cells* and this whole epidermal layer is named the *stratum spinosum* or *stratum acanthoticum* (Latin *spina* Greek *akantha* a spine thorn or 'prickle)

The *stratum germinativum* together with the stratum spinosum forms the part of the epidermis which is actually living and capable of reaction Therefore one frequently is in need of a word which encompasses both layers The terms *rete malpighii* rete mucosum or just rete for short are commonly used for this purpose More precisely it should be called the rete spinobasale. All rete cells are connected with one another by *epithelial fibers* (tonofibrils)

The *stratum granulosum* which lies just above the rete generally consists of one to two layers of flat and, in cross-section *spindle shaped cells* which are arranged parallel to the skin surface and contain rather large granules of *keratohyalin* This material is a cellular product associated with keratinization although it is not generally believed to be a direct precursor of keratin Its exact relation to keratin formation is not understood If the keratohyaline layer is very thick it may become visible through the horny layer as a bluish white substance This phenomenon causes the so-called Wickham s striae in lichen planus which will be described later

The *stratum lucidum* is a narrow light band between the granular and the horny layers It contains *eleidin* a protein derivative which like keratohyalin is associated with keratinization but its exact place in the process of keratin formation is not clear

The *stratum corneum* is the outermost layer of the epidermis. It is composed of compressed parallel homogeneous keratinized lamellae which no longer possess stainable nuclei The process of keratinization begins in the stratum spinosum where keratinizing protoplasmic fibrils pervade the intercellular bridges in basket like fashion From the top of the horny layer groups of keratinized and flattened cells are shed as microscopic scales a process which goes on unnoticed so to speak as insensible desquamation The smooth texture dull sheen and water resistance of the horny layer are qualities imparted by its cortert lipoid substances derived from keratinizing cells as well as from secrt from the sebaceous glands.

Between the epidermis and the cutis lies a *border layer* of fine interwoven fibers in whose meshes circulates epithelial lymph This layer is connected with the epidermis by footlike processes of the basal cells and with the cutis by fine collagenous and elastic connective tissue fibers.

like corkscrews through the epidermis, to end finally on the surface in tiny openings called *sweat pores* The sweat gland cells actively secrete sweat a watery acid fluid. All sweat glands are merocrine which means that their products consist of a secretion only and not of parts of cells. The cells of the sweat glands do not die in the process of secretion. The ordinary small sweat glands are called *eccrine* to indicate that no cellular protoplasm is shed with the secretion Besides these eccrine sweat glands, there exist in the axillary and pubic areas and also around the nipples larger *apocrine* coil glands which, like sebaceous glands, empty their secretion into the hair follicles. Apocrine sweat is mixed with fragments of protoplasm from the larger glandular cells and is a weakly acid to slightly alkaline milky secretion. Anatomically as well as functionally the mammary glands are related to the apocrine sweat glands.

The *hairs* and their follicles are implanted obliquely into the cutis. They are arranged in streams and whorls. The part of the hair which protrudes from the skin is called the *hair shaft* It has a roughly cylindrical shape and consists of long, spindle-shaped, horny fibers which according to the race contain more or less melanin. The hair shaft contains a main *cortex* and is covered by a *cuticle* whose scales are arranged like shingles on a roof The shafts of stronger hairs also contain central medullae That part of the hair which lies within the cutis is misleadingly called the hair *root* It is surrounded by the follicular sheaths, which on the surface continue into the epidermis and cutis. The hair root has a swelling at the base called the hair *bulb* Large hairs reach deep down into the cutis or even into the subcutis. The hair bulb which represents the generative tissue of the hair and contains many mitoses, overlies and partially envelops the hair papilla. This hair *papilla* consists of connective tissue and carries blood vessels from which the hair bulb derives its nourishment. On the average a hair grows almost 1.5 cm. (⅝ inch) per month or about 15 cm. (6 inches) per year After a certain period, which amounts to approximately 3 years for the long hairs, the hair separates from the bulb but still contains a club-shaped end (club hair) and is then finally expelled by a developing new hair Thus a continuous *change of all hairs is constantly* in progress.

The hair root is incased in the *hair follicle* which consists of several layers. The innermost layer is the *inner root sheath* a thin epidermal stratum of cells It forms a cuticular sheath, the cells of which tightly interlock with the cells of the hair cuticle It is, in turn surrounded by the *outer root sheath* which represents a continuation of the epidermal rete into the mouth of the hair follicle. This epidermal layer is coated by the *hyaline or vitreous layer* The whole epidermal structure is surrounded by a relatively dense connective tissue the *connective tissue hair follicle* in which the arrector pili muscle is anchored.

The arrectores pilorum muscles consist of smooth muscle fibers and, like the other appendages of the skin are sub-served by the autonomic nervous system The muscle fibers run from the uppermost layers of the cutis obliquely down to

system The autonomic *vegetative nerves* which are non-medullated run in the cutis there supplying the blood vessels the smooth muscles the glands and the hairs The *cerebrospinal nerves* ascend into the epidermis almost as far as the horny layer They are medullated but lose their medullae before entering the epithelium Some of these nerves terminate as free nerve fibrils while others form complicated terminal apparatuses which represent perceptive organs of sensation These include Merkel s tactile cells around the hair follicles and a whole series of different nerve corpuscles in the cutis, all of which have been named after their discoverers (Meissner Krause Ruffini Vater Paccini Golgi Mazzoni)

The *subcutis* consists of a tenuous network of connective tissues which carry blood vessels and nerves Its meshes are filled with grapelike clusters of fat tissue This skin layer is also *of variable thickness in different regions of the* body The thickness of this fat-containing layer obviously also depends to a large extent on the general nutritional status. This layer has manifold functions such as mechanical padding thermal insulation and storage of reserve food and water The rounding of body form which it accomplishes lends beauty to the female figure The amount of subcutaneous fat under given nutritional conditions is determined locally by influences intrinsic in the skin Thus abdominal skin if transplanted without fat to the dorsum or palm of the hand will develop subcutaneous fat tissue along with any general weight increase as if it were *still part of the paunch*

Appendages (adnexa) of the skin are oil and sweat glands hair and nails. They all are of ectodermal origin

The *sebaceous glands* (glandulae sebaceae) are situated in the upper part of the cutis and open mostly into a follicle thus forming in general a lateral annex of the hair follicle Generally where the gland is very large the associated hair and its follicle are small and vice versa Certain areas however have *free* sebaceous glands which have no connection with *the follicle These free glands* can be found in the vermilion border of the lips, in the inner laminae of the foreskin and in the labia minora The sebaceous glands are *acinous* (grapelike) in type having sacculated lobes They consist of large cells whose cytoplasm in cross-section *presents a honeycomb-like appearance* by a *process* of fatty degeneration The entire cells become converted into sebum Thus their secretion is of the holocrine type These glands *occur most numerously* on the nose and ears and along the anterior and posterior upper mid line of the body These latter areas also tend to accumulate sweat when it is *profuse and are* hence called the *sweat furrows* The palms and soles are devoid of sebaceous glands.

The *sweat glands* (glandulae sudoriferae) mostly lie deep in the cutis or even in the subcutis. They are fundamentally coiled double layered blind tubules Their long ducts run upward more or less vertically and then wind themselves

case the thickening may consist of normal keratin (*hyperkeratosis*) or of an abnormal keratin in which the cellular nuclei are retained and are stainable (*parakeratosis*). In the latter case the stratum granulosum, which is increased in hyperkeratosis, is usually lacking, as is also the stratum lucidum. Hyperkeratosis and parakeratosis may be seen side by side in the same histologic section. Another form of abnormal keratinization, called *dyskeratosis*, is characterized by premature formation of individual double-contoured, rounded, and enlarged keratinizing cells (*corps ronds*) and their shrunken end-product granules (grains). These three types of pathological keratinization will be more fully described later.

The horny layer may be thickened as well as loosened (scaling). It may be excessively saturated with sebum. It may contain pigment granules (in hypermelanotic conditions) or it may show accumulations of transmigrating leukocytes or their pyknotic nuclear residues (so-called micro-abscesses, especially in psoriasis).

If the stratum granulosum is thickened, as is usually the case in hyperkeratosis, the condition is called *granulosis*. In lichen planus, the granulosis is often so marked that the areas of increased accumulation of granulosum cells can be recognized with the naked eye as bluish-white little stripes and rings (Wickham's striae). Analogously to the terms *hyperkeratosis* and *granulosis*, a thickening of the rete, caused by an actual increase in rete cells and not merely by edema, is called *acanthosis*, so as to refer to the stratum acanthoticum or spinosum. In this condition the number of cellular layers is increased, and the rete ridges, therefore, may appear elongated. Acanthosis is encountered in a great variety of dermatoses, especially in those marked by inflammations. Conversely, the rete may be thinned, and the entire epidermis reduced to a very few layers of cells. In this case the rete ridges are usually absent, as is the case in scars (see p. 3 and Fig. 155). Intercellular edema in the epidermis may deform and push apart the epithelial cells to such a degree that the intercellular bridges tear, giving rise to microscopic vesicles which give the rete a spongy appearance (*spongiosis*). These microscopic vesicles may coalesce and form macroscopic vesicles (*vesiculae*). Vesicles may also be caused by *intracellular edema*, starting with unicellular vesicles which later coalesce (*altération cavitaire*) or by necrobiotic processes (*ballooning or reticular degenerations*). The significance of these different types of vesicle formation will be discussed later on page 44.

Of course, the rete cells may become *tumor cells* and send invading extensions into the structures beneath (epithelioma). In other instances, the entire epidermis or only one or more upper layers of it may be lacking (erosions, excoriation). In the stratum basale the number of mitoses may be increased or the amount of pigment in this layer may be greater or less than normal. In a considerable number of inflammatory skin diseases there occurs edematous destruction of the basal layer with removal of the pigment (mostly by pigment-laden migratory cells, called *melanophores*) into the cutis (*incontinentia pig*

the hair follicle so that their contraction raises the hair Their action becomes visible on the surface of the skin by the formation of transient little bulges which simulate papules a phenomenon generally known as goose flesh or cutis anserina. Contraction of these muscles also enhances the delivery of sebum Besides the muscles of the follicular apparatus one finds smooth muscle fibers in certain areas of the cutis (e g scrotum nipples) which form coherent muscular layers

There are various types of hairs, including the *long hairs* (scalp bearded area, axillae, pubes) the *bristle hairs* (eyebrows, lashes, vibrissae of the ear and nose openings) and the *downy hairs* (lanugo) which cover almost the entire body except the palms and soles, the fingertips and the foreskin In the fetus they form a dense growth of downy hairs (*primitive or fetal hair growth*) which however either sometime before or shortly after birth starts being shed. At the time of birth the development of the secondary (*permanent*) hair growth is already in progress, and fetal hairs are gradually replaced by terminal hairs. In the hair covering of infancy both types fetal and terminal are simultaneously present. At puberty terminal hair development is everywhere completed. The hairy covering then consists of the *diffuse hairy coat* which has replaced the fetal lanugo and the sexual hair growth which under the influence of the sex hormones appears only in certain regions (face axillae mons pubis)

The *nails* consist of the *nail plates* which are intimately connected with the epidermis of the *nail bed* (hyponychium) The nail plates are more or less convex smooth translucent and horny The nail bed has no part in the formation of the nail plate except for its most proximal portion the *matrix* which underlies the so-called nail root, the imbedded posterior edge of the plate. The matrix is actually the productive part of the nail bed from which originates the growth of the nail It extends from the proximal end of the nail to the distal border of the *lunula* a milky-colored crescent shaped zone of the nail which indicates the area where the nail plate is still connected with the matrix Posteriorly and laterally the edges of the nail plates are inserted into the *nail grooves* They are covered by the *nail fold* or *nail wall* The stratum corneum of the nail fold extends over the nail plate, forming the *nail cuticle* which gradually detaches itself from the growing nail At the free edge, the nail plate detaches itself from the nail bed. From the matrix, where it grows the nail plate moves distally at an even rate of almost 1 mm. per week or 3–4 mm per month

Pathological processes in the skin may of course, take place in various layers. Most of them are epidermo-cutaneous in nature so as to cause changes simultaneously in the epidermis and in the cutis.

EPIDERMIS

In the epidermis, either single or multiple layers may show pathologic changes. The stratum corneum may be thinned or thickened In the latter

(cm) The collagenous fibers may be subject to a variety of chemical degenerative changes which give rise to altered staining reactions (*collacin, collastin, and elacin degenerations*). Such degenerative processes which make the connective tissue flaccid and non-elastic, form the histopathologic basis of senile skin wrinkling.

SUBCUTIS

Pathologic changes in the subcutis play a part in but few dermatoses and in these *sclerotic processes*. *Wucher atrophy* of the fat tissue and *fat necrosis*.

In the *blood vessels* wall changes and thromboses, especially leukocytic ones, may be seen.

menti) This phenomenon is most marked in the pigmentary dermatosis of Siemens and Bloch

CUTIS

The *papillae* of the cutis may show dilated blood vessels or they may be extended with edema and shaped like mushrooms (lichen planus) In other conditions they are thinned elongated and finger like so that the adjoining rete ridges reach far down (psoriasis) Very frequently there is a *cellular infiltrate* in the cutis The infiltrate may be restricted to the stratum reticulare (papillae and subpapillary zone) ending sharply at its lower border Or it may extend far deeper with a marked tendency to follow blood vessels (*perivascular infiltrate*)

The types of *cells* forming the infiltrate may vary widely In many acute inflammations an infiltrate of *polymorphonuclear leukocytes* may be found. Under other circumstances especially in subacute and chronic inflammation *lymphocytes* may predominate Sometimes connective tissue cells which resemble epithelial cells (*epithelioid cells*) may accumulate and be surrounded by a fringe zone of lymphocytes while centrally there may be multinucleated *giant cells* of the Langhans type There may also be caseation This so-called tuberculoid structure is encountered not only in tuberculosis of the skin but also in a great variety of other granulomatous diseases *Eosinophils* may be increased in number or the picture may be dominated by *fibroblasts* and *histiocytes* Besides the Langhans type of giant cell other types are known including Dorothy Reed–Sternberg cells in Hodgkin s disease Touton giant cells in xanthomas foreign body giant cells nevus giant cells and epithelial giant cells such as occur in Bowen s disease In common intradermal nevi there occur *nevus cells* which represent another type of epithelioid cell believed to derive primordially from the embryonic neural crest In deeper chronic inflammations one frequently finds an increase in *plasma cells* a non-granulated type of leukocyte with an eccentric often cart wheel-shaped nucleus. In most inflammatory lesions and in the vicinity of tumors the so-called *mast cells* are increased This is most marked in urticaria pigmentosa which therefore may be looked upon as a mast cell granuloma The mast cells can be recognized by their coarse basophilic granules Certain macrocytic cells the so-called *melanophores* or chromatophores may gorge themselves with melanin pigment from the basal layer as in melanoses and the pigmentary dermatosis of Siemens and Bloch Accumulation of dermal melanocytes may also occur and give rise to visible blue lesions (Mongolian spot blue nevus) There may also be found *degenerated* cells and *cellular nuclei* distorted by pyknosis or karyorrhexis or *deposits* of blood blood pigment (hemosiderin) cholesterol (xanthoma cells) calcium amyloid mucin foreign bodies etc The cutis may also be invaded by *tumor cells* of variable types and origin

The connective tissue itself may undergo changes. The *elastic fibers* especially may be changed in their configuration or they may be scarce or entirely absent

GENERAL DIAGNOSIS

GENERAL DIAGNOSIS

Introduction

A PRIME characteristic of dermatologic diagnosis is that it is based on visual observation and that frequently despite extensive changes, *minute details* which can just be recognized with the naked eye are of the greatest importance. It is the *lesion* often a tiny alteration of skin, which forms the basic element of the most extensive skin eruptions. It is the observation of the lesion which leads to a clinical diagnosis. Such a scrutiny of minute things requires, like all conscious seeing effort and practice

What is the hardest of all.
It is what the simplest appears,
Just to see with your eyes
What lies in front of your eyes.

GOETHE

Therefore the dermatologist needs, above all, good *light* Changes in surface texture, such as glossiness, as well as delicate differences in level of lesions are best brought out by oblique or tangential lighting A magnifying glass is often indispensable. The examination must be made at *close range* A diagnosis made from a distance greater than 20 cm (8 inches) is, dermatologically speaking unsound long distance work.

A glance at the *history of dermatology* may serve to emphasize the decisive importance of the lesion in the diagnosis of skin diseases. Scientific dermatology was born in 1776 when Joseph Jakob Plenck of Vienna conceived the idea of *classifying the skin diseases according to lesions*

Collecting information is an early step in the development of any science Therefore the first two dermatologic textbooks consisted of a compilation of fact and information concerning skin diseases gleaned from Galen and a host of other ancient authors (Mercurialis 1571 Hafenreffer 1630) The first author who also contributed a fair amount of his own ideas for a dermatologic textbook was Lorry of Paris (1777) He lacked however a suitable viewpoint with which to co-ordinate his experiences into a coherent and logical system which is necessary if we want to pass on our experience to others or in other words, if we want to teach Without system, there is chaos. And it is chaos, hopeless disorder that we find in Lorry s book if we examine it in retrospect. For instance it is very difficult to make out from his description whether he is talking about acne or about freckles. Plenck s idea of constructing a system of skin

diseases based on lesions by first describing and precisely defining the various types changes the situation completely Single words often became sufficient to differentiate the disease without misunderstanding In the case of acne and freckles one could *simply speak of* 'pustules and 'pigmented spots.

Other dermatologists before and after Plenck had conceived other systems. Following Linnaeus (1707–78) it had almost become a fashion to classify diseases like animals and plants into genera species, and subspecies, sometimes with curious results The Plenck system however was not simply one among many *classifications.* In dermatology it was a critical milestone for physicians became compelled to look most consciously for *minute details* of the skin eruption if they wanted to put it into this system Thus Plenck became the great educator of physicians and taught them to *see dermatologically* and to pay attention to *every small and subtle detail* It was as if the magnifying glass had suddenly been invented and everyone began to see details which had been unknown before This became necessary because it was required by the system which proclaimed the law *No diagnosis of a skin disease without previous diagnosis of the lesion!*

Plenck s system was only an outline. The Englishman Willan and after his untimely death his disciple, Bateman wrote the *first textbook of dermatology* based on this system (1798 and 1813) It is significant that these two textbooks contained only a single table of illustrations, which simply showed pictures of the lesions *This fact well expresses the dominant importance of the lesions* as the foundation of dermatological teaching In comparison our modern texts mislead the student because they depict whole body regions the novice was easily led to believe from such pictures that the dermatologist arrives at his diagnosis in the same way as these pictures are made namely by observation *from a distance. On close examination the reader will find* that my book with its hundreds of pictures of lesions represents actually only a resumption and continuation of Plenck s idea but extended one hundred *fold* by using modern techniques Plenck s idea of the dominant importance of the lesion in the diagnosis of skin diseases is still alive and fertile, a manifestation of his creative *genius through the centuries.*

Bateman s text has had several editions, and for decades it was the most popular dermatological textbook in the world Later (1828) the book by Cazenave and Schedel (following Biett s lectures in Paris) gradually replaced it This book is also based on Plenck s lesion principle and it also contains only one table of illustrations but differs from the Willan Bateman text in that all lesions are inserted in a human figure

It is true that *Alibert* of *Paris an outstanding teacher of* dermatology tried to free himself from the Plenckian system (1806 and 1825) However his classification of skin diseases was so confused and impractical *that it had no* followers nd soon fell into oblivion For example in Alibert s book such

heterogeneous ailments as ichthyosis, dermatolysis, verrucae, nail diseases, and nevi are grouped together as "dermatoses heteromorphae" and favus and pediculosis (plica polonica) as "dermatoses tineosae."

Another great mistake in the classification of skin diseases prior to Plenck was the placing of undue emphasis on distribution as a leading consideration in diagnosis. This approach inevitably fosters superficiality since the distribution can be recognized without the slightest attention to details. Thus Alibert tried to maintain as fundamentally different the "teignes" (tineae) of the scalp from the "dartres" (desquamations) on the rest of the body. Consequently—and unfortunately—the same disease could be labeled with different names, depending on the site where the lesions were located.

In the mid-nineteenth century, efforts to create a system in dermatology followed new paths. The second great advance in the development of scientific dermatology came when Ferdinand Hebra of Vienna made *pathologic anatomy*, which then flourished under Rokitanski's leadership, the foundation of dermatologic observation and of a new classification of skin diseases. The contrast with Plenck's ideas was only a superficial one, since histopathologic features are reflected clinically in *lesions*, which Hebra thus retained as the starting point of dermatologic diagnosis. Finally, at the turn of the century, the *etiologic* conception gradually gained prominence so that Darier, of Paris, for the first time undertook to classify the diseases in a dermatologic text *according to their causes* (J. Darier, *Précis de dermatologie* [Paris: Masson & Cie, 1908]). Because this etiologic classification was possible only for half the entities, he felt it necessary to write, in addition to the "Nosology of Dermatoses," a voluminous "Morphology of Dermatoses." The result was that he had two texts of equal size, each comprising half the domain, which had to be glued together. The "morphologic" portion, which precedes the nosologic one, is again based entirely on the lesion plan. This is easy to understand because the etiologically defined dermatoses as well as the others can be recognized only by their morphology, which is nothing but the lesions. From all these developments the student can derive the following moral: He who studies skin diseases and fails to study the lesion first will never learn dermatology.

Thus, even in modern textbooks, the very foundation of dermatologic examination and diagnosis remains the lesion. Lesions are the alphabet without which nobody can read the language of the skin (Darier). The morphology of the lesions alone is able to suggest a preliminary idea of the pathologic anatomy and etiology until histology, bacteriology, and history provide full confirmation of the diagnosis, if necessary. The fundamental importance of the lesion should not, of course, close our eyes to the fact that some other data may be of significance for the dermatologic diagnosis. For the beginner and also for the experienced in difficult cases, it is best to gather all the facts into a complete "present status," as is customary in internal medicine. In this way nothing

will be overlooked which has to be evaluated even if it is only of minimal significance

The *color* of a skin change is the first thing to be considered because it produces the first impression Second the surface must be studied in order to determine the *level* of the altered skin in relation to the normal surrounding skin This observation is related to the recognition of the *lesions* which make up the eruption in question or the *deposits* which cover the lesion such as a crust or an erosion or ulcer Following this, *consistency* (the hardness or softness) of the changed skin is examined and at the same time information about its extension downward into the invisible layers is obtained These characteristics may be called *signs of the first order* on which a sound diagnosis must be based In certain cases there exist other important characteristics which can not be obtained by the observation of local details. Among these are the extent and distribution of the eruption the arrangement of the lesion and the nature of the border of the eruption The appendages (hair nails) and the mucosae must be examined and systemic signs and general symptoms (fever pallor emaciation) as well as symptoms pointing to other organs should be watched. Finally the case workup is completed by questioning the patient especially about the duration and course of his illness as well as his subjective symptoms (itching burning pain)

In short determination of the clinical dermatologic status rests on observations and information on the following points (1) *color* (2) *lesion* (level deposit) (3) *distribution* (arrangement site) (4) *Consistency* and depth of extension (5) *hair nails mucosae* (6) *systemic signs and symptoms* (symptoms and signs caused by organs other than the skin) and (7) *subjective symptoms* and history of the case

To secure information about the distribution and extent of the eruption ideally the entire skin surface should be examined. This is also often necessary for the specific diagnosis of *lesions* which may undergo considerable changes during their existence and lose characteristic features One therefore has to search for *primary types of lesions* i e lesions in their early stages of development Such lesions however may be situated in places which are covered by clothing and the patient may think that the examination of just these sites is unimportant Experience teaches us repeatedly never to believe the assurances of a patient that there is nothing on the covered parts of the body A complete dermatologic examination often has to include certain regions of the body which people may dislike to show It is thus often necessary to inspect not only the scalp the oral mucosae the palms and the nails but also the genitalia the anus, and the feet In all these areas skin changes may be found which can unexpectedly aid in making the diagnosis. Sometimes incidental findings may also be revealed the oversight of which might later be blamed on the examining doctor If possible the dermatologist should not depart from the rule of

examining the patient's entire skin at the first visit, if he wishes to be considered a conscientious examiner. This is an ideal and theoretically well-founded rule. Unfortunately it is not advisable to follow it too rigidly in private practice. A considerable percentage of the skin cases in private practice are acne and vesicular eruptions of the hands. Especially in female patients but also in men in such clearly recognizable cases it is better to run the slight risk of overlooking something than to lose the patient or get the reputation of being odd. The experienced dermatologist will very soon sense the cases in which it is undesirable and not strictly necessary to examine the patient entirely disrobed.

CHAPTER ONE

The Color

One can deduce many things from the color of a skin eruption which is often the first visual impression. The color of the skin is composed of *pigments* deposited in the skin and of the color of the *blood* which fills the cutaneous vessels. Occasionally the color of *cellular accumulations cellular products* and also *surface deposits* may play a part. Accordingly we have to discuss *pigments* (a) natural body pigments (skin melanin pigment carotene, bile pigment) (b) foreign pigments (tattoo drugs) *blood* in the vessels *cells* (a) the body's own cells, cellular fibers, and other cellular products, (b) foreign cells (fungi)

Melanin is the most important among the naturally produced body pigments and consists of almost iron-free, yellow brown to practically black granules which accumulate in the cells of the basal layer of the epidermis in the form of supranuclear caps. From here they are either carried away into the cutis by melanophores or shed from the surface with the insensible desquamation. They give the skin its tan to black (Negroes, morbus addisonii) or yellow (Mongolians) color Localized increase of melanin pigment such as occurs in freckles, chloasma gravidarum and certain nevi (Fig 1) is well known. In inflammatory processes, hyperpigmentation may already occur in the eruptive stages of disorders (secondary syphilis, lepra maculosa) It is, however a very common residual finding after healed inflammations, especially in brunet persons (e.g after impetigo eczema, toxicoderma, psoriasis, lichen planus, Figs. 2-4) In a sense hyperpigmentation can be considered the equivalent of a scar after superficial injuries (excoriations, burns) which heal without a real scarring I call these *secondary* postinflammatory increases in melanin pigmentation *melanodermas* to contrast with the opposite change called *leukoderma* which designates secondary decrease or loss of pigment following inflammation. It would be appropriate t call the endogenous and *primary* hyperpigmentations which develop without preceding skin disease *melanoses* The pigmentations in Addison's disease and in pregnancy are examples of melanoses, in this stricter sense. Pigment formation is enhanced by arsenic medication, especially in dark skinned persons.

The brown color f melanin becomes visible on the surface only if it is situated in the epidermis. Pigment that accumulates in the *cutis* in melanophores



Fig 1 —Hyperpigmentation (nevus pigmentosus spilus)



Fig. 2 —Melanoderma meaning secondary pigmentation, after papular eczema

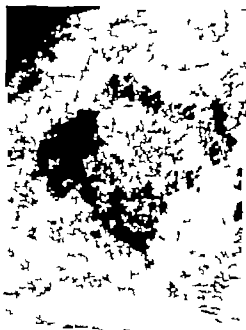


Fig 3 —Central melanoderma (vesicular dermatitis herpetiformis Duhring)

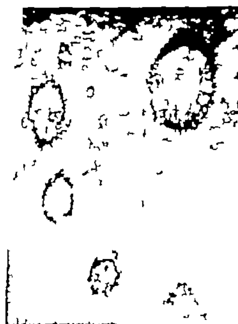


Fig 4 —Annular melanoderma (psoriasis)

acts like a dark background which appears blue if seen through the cloudy but still translucent skin on top of it. This is the same optical phenomenon as that which causes the iris of the eye which contains very little pigment, and also the sky to appear blue. Such blue spots due to accumulation of pigment in the deeper layer of the skin are encountered in the sacral area of the newborn, especially of the yellow race and are called "Mongolian spots." Blue is also found in certain nevi (nevi caeruleae) and in the pigmentary dermatosis of Siemens and Bloch.

Abnormal color of the skin may of course, also result from a *decrease in pigmentation* There is lack of melanin pigment in universal distribution in albinism. Absence of pigment in circumscribed areas is the essential finding in the piebald, in vitiligo (Fig 5) and in nevi depigmentosi. Lack of pigment like an excess of pigment may occur as a "scar equivalent" after superficial injuries to the skin. Transient depigmentation may also follow a variety of dermatoses, such as syphilis, psoriasis, lichenified eczema, etc. *Secondary* depigmentations are called *leukodermas* (Fig 6) In these conditions—as is sometimes the case in primary depigmentations—the pigment is only diminished and not entirely absent (Fig 7) The leukodermas should not be confused with such areas of normal skin as in the case of widely distributed eruptions, have remained clear (Fig 8) and conversely the remnants of normally pigmented skin in generalized depigmentations should not be mistaken for hyperpigmentations (Fig 9) Frequently hyperpigmented and depigmented areas are found close together (in scars, in local albinism, Fig 10) Small white spots may occur in hyperpigmented skin (sun tan arsenical melanosis, Fig 11) or depigmented spots may have brown borders (vitiligo various leukodermas) These combinations are known as *leukomelanodermas* or *shifts of pigment* One should however not be led to believe from this expression that an actual horizontal "shift" of the pigment has taken place.

When blood escapes from its vessels, as in a bruise there develops a deposit of yellow brown pigment, *hemosiderin* derived from hemoglobin. By virtue of its iron content, hemosiderin pigment can be differentiated from melanin with suitable histochemical tests. Bleeding arises in the skin in the vascularized cutis and is almost always confined to the cutis The macules which follow the extravasation are called *purpura* or *purpuric spots* They are slowly absorbed and disappear after days or weeks, depending on their size Purpuric spots may be punctate or very small (petechiae Fig 12) coin-size (suggillations) or very extensive (ecchymoses) At first, all superficial extravasations show the dark red color of blood Then gradually by decomposition and absorption of the hemosiderin, they turn brownish and yellowish. If they are situated fairly deep below the surface they show the same blue color that any dark pigment so situated assumes when viewed from the surface through the translucence of the overlying skin. This intense dark-blue color of a deep cutaneous blood deposit gives way



Fig 1 —Hyperpigmentation (*nevus pigmentosus spilus*)



Fig. 2 —Melanoderma meaning secondary pigmentation, after papular eczema

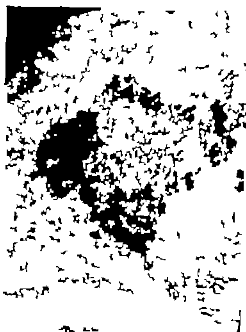


Fig 3 —Central melanoderma (edcula dermatis herpetiformis Duhring)



Fig 4 —Annula melanoderma (psoriasis)



Fig. 9 — Remnant of normal skin (*dark*) surrounded by vitiligo (*light*)



Fig. 10 — Depigmentation and hyperpigmentation closely adjoining (circumscribed albinism)



Fig. 11 — Leukomelanoderma after prolonged arsenical medication



FIG 5.—Pigment-free spot Primary depigmentation (vitiligo)



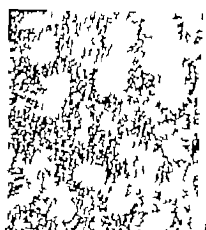


FIG 6.—*A* psoriasis and leukoderma *B* secondary depigmentation after clearing of psoriasis.



FIG. 7.—Central leukoderma (squamous eczema)



FIG. 8.—Unaffected skin (*light*) surrounded by dark areas of tinea versicolor

(argyria or argyrosis) may develop. Atabrine may stain the entire skin yellow The bites of pubic lice may cause lenticular slate-blue spots which have diagnostic significance (maculae coeruleae or caeruleae) Medicaments may of course also stain the skin *from without* This discoloration is very characteristic of numerous dermatologic medications, such as tars, chrysarobin, and silver nitrate.

Besides these various pigments, the blood within vessels is another main component that determines skin color Therefore, the normal skin has pink to purple as well as yellow to brown or black hues. The color contributed by the blood is more marked when the horny and keratohyaline layers are thin or en



Fig. 13 —Diascopy (glass pressure) of vascular nevus. The pressure is insufficient, so that some small blood vessels still remain visible

tirely absent. For this reason abrasions and the bases of blisters, as well as mucosae and the vermilion borders of the lips, are redder than the other parts of the skin The most significant factor causing skin redness is the *dilatation of blood vessels* (hyperemia) The capillaries of the uppermost layers of the cutis and even more the network of the small superficial veins play primary roles in this manner

In contrast t the coloration caused by fixed pigments, the color contributed by intravascular blood can be *removed by pressure* This is of diagnostic importance For demonstrating the elimination of this color by pressure, a small disk or spatula of thick glass is used a method known as *diascopy* Strong and prolonged pressure is som times necessary to compress the vessels without leaving some branches visible (Fig 13) When possible it is best to select a site

to greenish and yellowish hues as the blood pigment becomes altered and resorbed Purpuric spots besides manifesting hematologic or other systemic disease may occur as a harmless accompaniment of some skin diseases. On the lower legs of old people a great variety of inflammatory skin diseases (*eczemas* psoriasis ulcerations boils) are frequently associated with extravasations of blood. Purpuric macules may develop into palpable lesions or form blood filled vesicles (purpura papulosa purpura vesiculosa)

In a variety of pathologic conditions large amounts of *bile pigment* may enter the blood circulation and from there reach the skin and mucosae (conjunctivae) causing a characteristic yellowing (icterus)

The normal *pigment of the fat* (carotene or xanthophyll) may become visible as a yellow discoloration of the skin In the xanthosis of small children who are



Fig. 12.—Purpuric spots (petechiae)

overfed with carrots or spinach very large areas of the skin may become yellow Localized yellowish areas may be caused by accumulations of lipid or lipid like masses in or under the translucent skin (superficial follicular cysts, *enlarged sebaceous glands xanthomas) Changes in the color of the skin may* also be caused by *foreign pigments* Best known are the blue patterns of tattoos which are produced by the introduction of black India ink into the cutis. The blue color results from the same optical phenomenon as that which causes deep-seated skin pigment and blood extravasations to appear blue For the same reason green patterns evolve from the introduction of blue particles of pigment Cinnabar and carmine are used to produce red tattoos Unintentional tattoos may be caused by powder particles from gunshots fired at close range or by abrasions acquired in coal yards. Similar occupational tattoos of grinders stone masons, miners, etc may follow the entrance of steel quartz or coal particles into the skin Medicaments may also cause cutaneous or mucosal pigmentations. Bismuth injections may cause a bluish gray deposit of *bismuth sulfide* at the edges of the gums. After prolonged treatment with silver compounds such as silver nitrate a diffuse bluish slate-colored discoloration from silver particles

vulgaris. The temperature of inflamed skin is increased. Erythema is often the longest lasting sign of a dermatitis and may not vanish for weeks or months. This phenomenon is called *residual erythema* (Fig 15) The vasomotor ery themas may be caused by *active* as well as *passive hyperemias* (flush hyperemia and stasis hyperemia) Emotional erythema (erythema pudoris) and the flushed face of the febrile—also the hectic flush—are representative of the first group The *passive hyperemias* are produced by congestion in the small veins or capillaries of the skin. Their characteristic color is a darker more bluish red. Some authors avoid the term *erythema* preferring to speak of *cyanosis* or *livedo* It would be logical to speak of a *cyanema* (*cyanos* Greek for "blue) and a



Fig 16 —Reticulated passive erythema (cutis marmorata congenita)

leukanthema (*leukos* Greek for "white") as opposed to the hyperemic *erythema* (*erythraios* Greek for red) Stasis, however may on occasion produce bright red spots, such as the so-called cinnabar spots on the dorsa of the hands in acroasphyxia

In contrast to the warmth of active hyperemia cyanotic skin generally feels cool, even though there are some exceptions to this rule such as the 'warm stasis artificially produced with a tourniquet. Anemic spots which have been produced in cyanotic skin by finger pressure lose their white color slowly It is easy to understand why the acral parts (prominent and dependent parts, including nose ears, hands, feet etc) have a marked tendency toward stagnation and cyanosis.

A peculiar type of congestive hyperemia is the net-shaped livid discoloration known as *cutis marmorata* or *livedo annularis* (Fig 16) The arrangement of the

where the glass can be pressed against an underlying bone Sometimes, even with this maneuver a few red puncta may remain because of the presence of cystically dilated or coiled blood vessels whose outlets as well as inlets, are likely to be shut off by pressure (papular telangiectases)

Hyperemia may be *inflammatory* or *vasomotor* In either case it is more or less transient and produces a type of redness called *erythema* (Fig 14) Ery-

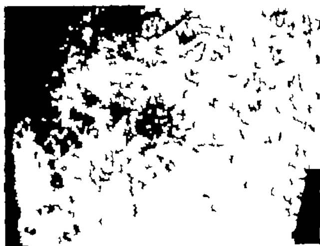


Fig 14 —Erythema (eczema erythematosum)



Fig 15 —Residual erythema after impetigo

thema is a recognized cardinal sign of *inflammation* The ancients had already coined the formula *rubor* calor dolor tumor (redness heat pain swelling) for the main features of inflammation In acute conditions the inflammatory hyperemia is bright red whereas in chronic conditions it is more livid and often mixed with brown so that a dirty red or ham color results. The brown component arises from deposition of brown pigments or the accumulation of certain cellular infiltrates such as occur in secondary and tertiary syphilis and lupus



Fig. 17.—Macular telangiectases at the nape of the neck



Fig. 18.—Branching telangiectases in the early stage of rosacea of the nose



Fig. 19.—Papular telangiectases (angiomata senilia, ruby points)



Fig. 20.—Papuloramal telangiectases (so-called nevus araneus, vascular spider)

network in this condition reflects the vascular supply of the skin the normal colored centers corresponding to the areas of direct blood supply from an arteriole while the livid network itself represents the shunt zones where the circulation even under normal conditions is relatively slower Cutis marmorata occurs in different forms cutis marmorata *vascularis* (annularis or reticularis) is the most common one It occurs in many persons on disrobing and is then called cutis marmorata *vascularis e frigore* (caused by cold) There is also a permanent type of this anomaly called cutis marmorata *perstans* In some cases the livid meshwork is caused by inflammation cutis marmorata *inflammatoria* or *racemosa* (from *racemus* Latin for cluster of grapes not a very apt comparison) Its manifestations are persistent of course and stable There are also ramifications besides the rings and the livid meshes may show slight thickenings which may even become lichenified The condition occurs in circulatory disorders and infectious diseases such as syphilis and tuberculosis, but it may also be congenital without any recognizable cause. The same picture with deep-seated little nodules of phlebitic or other nature is known as cutis marmorata *nodosa* and occurs especially in periarteritis nodosa together with the other symptoms of the disease (weakness and anemia polyneuritis myositis and gastrointestinal disturbances) Occasionally there is hyperpigmentation in the network so that the receding cyanosis leaves a net shaped melanoderma, called cutis marmorata *pigmentosa* (pigmentatio reticularis) Occasionally there may be blood extravasations causing deposits of hemosiderin in addition to melanin. Such marble-patterned melanodermas occur most frequently after localized and prolonged applications of heat and are called cutis marmorata e calore (e g after hot compresses on the abdomen or after exposing the legs to the heat of a stove) This pigmented type of cutis marmorata may also be congenital

If the redness of the skin is not caused by congested blood vessels but rather by a permanent enlargement in caliber coiling and increase in number of the vessels of the skin the lesion is no longer called *erythema* but rather *telangiectasis* or *telangiectasia* Such reddening of the skin may be evenly mottled (*macular* telangiectases Fig 17) or on close inspection it may prove to be composed of fine linear branches of blood vessels (*ramal* telangiectases Fig 18) Both types are frequently found in so-called ruddy cheeks.

More deeply seated telangiectases become visible as livid to purplish streak like macular branches. They are often arranged in bunches as in the vascular garland on the costal arch or the vascular bunches on the thighs and lower legs If the dilated cutaneous vessels are entwined in small balls they become visible as dark-red ruby points and nodules (*papular telangiectases* so-called *angiomata senilia* Fig 19) In the spider nevus (nevus araneus or more correctly telangiectasis aranea) the vascular branches radiate from a raised papular center forming a *papuloramal* telangiectasis (Fig 20) All three types of tel



Fig. 17.—Macular telangiectases in the nape of the neck.



Fig. 18.—Branching telangiectases in the early stage of rosacea of the nose.



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yellow color of horny substance (callus, clavus, verruca, lichen planus) which color also becomes visible in dried blister tops (Fig. 24). After a prolonged time keratinized cells take on a greenish-black color because of oxidation (e.g., follicular keratoses, Darier's disease, ichthyosis, Fig. 25). Thus gray-brown or black "pseudo-pigmentation" of the horny layer which is not caused by melanin but is actually the color of the cells after dehydration and oxidation, is called



FIG. 22.—Anemic spot without (*A*) and with (*B*) glass pressure (nevus anemicus).

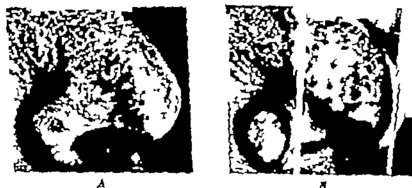


FIG. 23.—Lupus nodule without (*A*) and with (*B*) glass pressure.

phaeoderma (phaios, Greek for "gray brownish"). Besides leukoderma, phaeoderma is another color change to contrast with melanoderma. With sufficiently close observation, it can easily be differentiated from the latter.

The blackening of horny masses may also be caused by their saturation with dirt, as, for example, the cracks of calluses on the hands and feet. Chemical oxidation of horny substance saturated with sebum causes the black color of the tips of comedones. If horny layers are loosely held together, air easily

angiectases—macular ramal and papular—may be combined in vascular nevi (nevi flammei strawberry marks) If larger vessels are involved they take on a dark purple color Of course larger veins running in the subcutis appear blue through the skin. This is normally the case on the temples and forearm flexures but it becomes especially distinct if the skin is thinned, as in all atrophies or if the veins are much dilated (varicose veins)

Just as the skin becomes darker and redder by vasodilatation so it becomes lighter and paler by *constriction of the blood vessels* or reduction of their numbers (anemia) Everybody knows this phenomenon from the vasomotor paleness associated with fright and fear (emotional pallor anemia pavoris) Circumscribed anemia makes light spots which can usually be differentiated from depigmented spots only by diascopy (Figs. 21 and 22) Since they are small it cannot as a rule be verified that their temperature is below normal The light



Fig. 21 —Depigmented spot (nevus depigmentosus)

shade of older scars can be explained by a combination of anemia and depigmentation Anemic light spots *without* other skin changes are encountered in nevus anemicus. Some skin lesions have light-colored anemic halos.

The yellowish color of *serum* like the color of the blood may become noticeable if it leaks from the vessels as in eczemas accompanied by edema If moderate glass pressure is applied to lesions of this kind, the inflammatory dilatation of the larger vessels vanishes and the amber color of the serum becomes visible

Cells and cellular products besides pigments and blood, may also determine the color of pathologic changes in the skin Thus the conglomerations of lymphocytes, giant cells, and epithelioid cells which occur in skin tuberculosis appear under glass pressure as dusky amber or grayish brown spots. This is of great importance in the diagnosis of skin tuberculosis (Fig 23) On the other hand the red spots of lupus erythematosus the papules of eczema and wheals disappear completely under glass pressure *Keratinized cells* have the brown

yellow color of horny substance (callus, clavus, verruca lichen planus) which color also becomes visible in dried blister tops (Fig 24) After a prolonged time, keratinized cells take on a greenish-black color because of oxidation (e.g follicular keratoses, Darier's disease ichthyosis, Fig 25) This gray brown or black "pseudo-pigmentation of the horny layer which is not caused by melanin but is actually the color of the cells after dehydration and oxidation is called

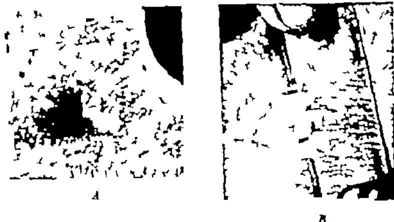


Fig. 22 —Anemic spot without (A) and with (B) glass pressure (nevus anemicus)



Fig 23 —Lupus nodule without (A) and with (B) glass pressure

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angiectases—macular ramal and papular—may be combined in vascular nevi (nevi flammei strawberry marks) If larger vessels are involved they take on a dark-purple color Of course larger veins running in the subcutis appear blue through the skin This is normally the case on the temples and forearm flexures but it becomes especially distinct if the skin is thinned as in all atrophies or if the veins are much dilated (varicose veins)

Just as the skin becomes darker and redder by vasodilatation so it becomes lighter and paler by *constriction of the blood vessels* or reduction of their numbers (anemia) Everybody knows this phenomenon from the vasomotor paleness associated with fright and fear (emotional pallor anemia pavoris) Circumscribed anemia makes light spots which can usually be differentiated from depigmented spots only by diascopy (Figs 21 and 22) Since they are small it cannot, as a rule, be verified that their temperature is below normal The light



FIG 21 —Depigmented spot (nevus depigmentosus)

shade of older scars can be explained by a combination of anemia and depigmentation Anemic light spots *without* other skin changes are encountered in nevus anemicus Some skin lesions have light-colored anemic halos.

The yellowish color of *serum* like the color of the blood may become noticeable if it leaks from the vessels, as in eczemas accompanied by edema If moderate glass pressure is applied to lesions of this kind the inflammatory dilatation of the larger vessels vanishes, and the amber color of the serum becomes visible

Cells and cellular products besides pigments and blood may also determine the color of pathologic changes in the skin Thus the conglomerations of lymphocytes giant cells and epithelioid cells which occur in skin tuberculosis appear under glass pressure as dusky amber or grayish brown spots. This is of great importance in the diagnosis of skin tuberculosis (Fig 23) On the other hand the red spots of lupus erythematosus the papules of eczema and wheals disappear completely under glass pressure. *Keratinized cells* have the brown

yellow color of horny substance (callus, clavus, verruca, lichen planus) which color also becomes visible in dried blister tops (Fig. 24). After a prolonged time keratinized cells take on a greenish-black color because of oxidation (e.g. follicular keratoses, Darier's disease, ichthyosis, Fig. 25). This gray-brown or black "pseudo-pigmentation" of the horny layer which is not caused by melanin but is actually the color of the cells after dehydration and oxidation is called

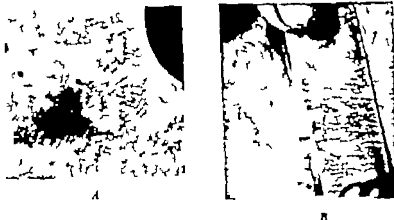


Fig. 22.—Anemic spot without (*A*) and with (*B*) glass pressure (nevus anemicus).

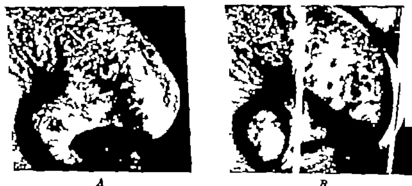


Fig. 23.—Lupus nodule without (*A*) and with (*B*) glass pressure.

phaeoderma (*phaios*, Greek for "gray brownish"). Besides leukoderma, phaeoderma is another color change to contrast with melanoderma. With sufficiently close observation it can easily be differentiated from the latter.

The blackening of horny masses may also be caused by their saturation with dirt, as, for example, the cracks of calluses on the hands and feet. Chemical oxidation of horny substance saturated with sebum causes the black color of the tips of comedones. If horny layers are loosely held together, air easily

enters and causes a silvery appearance by reflection of light from the separated horny lamellae (pityriasis simplex psoriasis) If the horny layer is soaked and waterlogged it is thrown into coarse folds and finally becomes turbid and white (washerwoman s hand macerated skin) *On the mucosae which normally* lack a horny layer maceration of the upper epidermal layers may create the same milky turbidity (plaques opalines in syphilis) while adherent, persistent



A



B

FIG 24.—Phaeoderma *A* grayish-brown discoloration of a dried blister top (Stevens-Johnson syndrome) *B* at right blister top has been lifted and turned t show light-colored blister floo

masses of (pathological) keratinous layers tend to take on a yellow hue (so-called leukoplakia actually keratoses mucosae) A whitish milky appearance with more bluish hues may be created by the thickening of the keratohyalin containing granular layer (Wickham s striae in lichen planus and lichen planus of the mucous membrane)

The connective tissue *intercellular substance* may also become visible in lesions. Thus non-pigmented and scantily vascularized scars show the white color of the connective tissue of the cutis. If however the elastic tissue of the cutis has degenerated in a certain way it may become visible and show a distinct yellow color (pseudo-xanthoma elasticum connective tissue degeneration in some scars) If the skin is very thin (senile atrophy acrodermatitis chronica atrophicans) the yellow tendons may be readily visible along with the prominent blue veins.

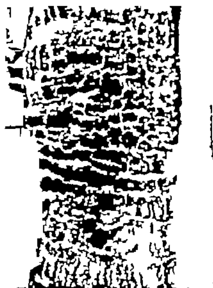


Fig 25 —Phacoderma, black keratotic scales in ichthyosis congenita

Finally the color of lesions may be influenced by *foreign cells* which are attached to the skin externally This is the case in superficial fungus diseases and is exemplified by the mealy white scales of microsporia the lemon yellow accumulations of favus, known as scutula (*scutulum* Latin for "little shield") and by tinea versicolor in which the dried-out horny layer with the dense brown mycelia of *Malassezia furfur* may be mistaken for hyperpigmentation

CHAPTER TWO

The Lesions

The second and, for dermatological appraisals most important considera tion is the *lesion* As a teacher I cannot see much advantage in the usual division into primary and secondary lesions It is more important always to start an examination by determining the level of the lesions in relation to that of the normal skin It is of practical value to distinguish lesions *in above* or *below* the level of normal skin This then should be followed by the description of the scales and crusts which are usually classified in the category of second ary' lesions but which should rather be grouped separately as *deposits* since they are non living and can be detached from the skin

The systematic discussion of lesions will be taken up in the following order

In the plane of the skin.
- *Macula* (macule, spot)

Transitional not restricted only to this plane.
- *Erythema* (redness)
- *Telangiectasis*

Raised.
- *Urtica* (wheal pomphus)
- *Vesicula* (small blister vesicle)
- *Bulla* (blister)
- *Cyst*
- *Pustula* (pustule pus vesicle)
- *Abscess*
- *Papula* (papule nodule)
- *Tuber*
- *Node*
- *Tumor* (growth)
- *Vegetation*

Transitional, not restricted to any one plane.
- *Cicatrix* (scar) hypertrophic and atrophic

Depressed.
- *Atrophy* (thinning of the skin)
- *Anetoderma*
- *Erosion*
- *Excoriation*
- *Wound*
- *Ulcer*

Deposits (scoriae)
- *Scale*
- *Crusts*
- *Foreign cells and cell products*
- *Dirt*

Before discussing these lesions one after another we should consider those skin disorders which lack signs. There are in this category various forms of itching such as pruritus senilis or *itching* associated with jaundice nephritis carcinoma hypertrophy of the prostate, or local stasis (varicose veins, hemorrhoids) Also certain epizoa (pinworms, pubic lice) may cause itching without visible skin lesions. Most types of pruritus, however sooner or later lead to the production of erythematous streaks, erosions excoriations (scratch effects) dermatitis (eczema) and even pyoderma by bacterial infection of the scratch wounds.

A *macule* (spot) is a circumscribed deviation from normal skin color without other changes, especially without alteration of the surface (glossiness dulness, surface relief) of the level, or of the consistency of the skin. Blue spots (maculae caeruleae) may give the impression of being below the level of the skin. One should be on guard against this error Macules may vary in size from a point or a small speck to large areas. The borders of macules may be distinct or vague, and their shapes although extremely variable may sometimes suggest the mode of their development as is the case in other types of lesions. Round and oval shapes suggest a peripherally growing process combinations of convex arcs (polycyclic gyrate) can be interpreted as resulting from the confluence of several individual lesions. Geographic maplike spots with jagged edges are encountered almost exclusively in stationary skin lesions (birthmarks) Rectangular edges and generally all odd-shaped lesions suggest artifacts. However linear or stripe-shaped lesions occasionally occur in inflammatory dermatoses and in congenital malformations (systematized eczemas systematized nevi)

The anatomical basis of a macule may consist of any of the changes which we have mentioned in discussing discolorations of the skin. Thus the *color* of macules is extremely variable. Different degrees of translucency cause further divergences, which have given rise to descriptive expressions, such as "apple jelly-like (lupus nodule) or "honey-like" (impetigo crust) Red is the most common color (erythema and telangiectases) Brown occurs in all possible shades and hues. Blue, yellow white and black are frequently observed In the majority of cases we are dealing with mixed colors, which is why we customarily use such terms as bluish white, "livid red, "ham red," "yellowish brown "bluish brown, etc It is because of this fact that the color of most macules and, for that matter of other lesions consists of different anatomical components. For instance the color of certain drug eruptions, older luetic lesions, and leprotic macules results from hyperemia as well as hyperpigmentation. The color of pityriasis versicolor comes from the color of the drying horny layer and the color of the masses of fungus elements. Not infrequently the true color of macules is masked by accompanying hyperemia. This, of course is also true of other types of lesions, particularly inflammatory ones. In this case the true color of macules becomes visible only after the dilated blood vessels have been

compressed by a glass plate (diascope) Diascopy is also necessary for the accurate differentiation of spots caused by *intravascular* and *extravascular* blood that is of erythema and telangiectases on the one hand and purpura on the other This situation can be well illustrated by fresh fleabites. These consist of bright red oval spots with dark red points in their centers (Fig 26) Under glass pressure the red spots disappear while the dark centers persist (Fig 27) Thus we are dealing with lenticular (lentil sized) erythema with central punctiform purpura.

Diascopy is a useful method not only for differentiating hyperemia and hemorrhage but also *for distinguishing anemia and depigmentation* in light colored macules The difference here can be demonstrated most clearly if pressure is applied at the border between the discolored and the normal skin If the discoloration is caused by differences in vascularity across the two sides



Fig 26.—Erythema with central hemorrhage (fleabite)

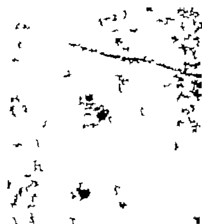


Fig 27 —Same under glass pressure hemorrhage stays, erythema fades

of the border the border line will disappear under the glass (Fig 28) while in the case of depigmentation or hyperpigmentation it will remain unchanged (Fig 29)

The following outline shows the most important causes of the *various colors* of macules and other lesions

Red —Hyperemia (inflammatory or vasomotor) telangiectases, fresh superficial cutaneous hemorrhages (purpura)

Brown —Skin pigment (hyperpigmentation melanoderma) decomposed blood pigment (purpura in clearing) chronic inflammatory cellular infiltrates (lupus vulgaris, syphilis) keratinized cells (calli, clavi) fungus growth (pityriasis versicolor) dried serum (crusts of impetigo)

Blue —Deep-seated pigment (Mongolian spot, blue nevus) deep-seated blood pigment (purpura) deep-seated black dyestuff pigments (tattoo) deep-seated dilated blood vessels (hemangioma) spots caused by the bite of crab lice (maculae caeruleae)

Green —Deep-seated decomposed blood pigment (purpura profunda in clearing) pus (yellowish green)

Yellow.—Skin pigment (Mongolian race) blood pigment (purpura in its last phase) bile pigments (icterus) lipoid pigments (xanthoma, sebaceous cysts) keratinized cells (callus, clavus, cornu cutaneum) degenerated connective tissue (pseudo-xanthoma) pus (pustules, crusts) fungus growth (favus)

White.—Absence of skin pigment (albinism, vitiligo leukoderma) absence of blood (anemia) thickening of the stratum granulosum (Wickham's striae in lichen planus) waterlogged (macerated) epidermal and especially horny cells (plaques opalines, leukoplakia) horny cells interspersed with air (loose scales, especially in psoriasis) fibrinous pseudo-membranes on mucous membranes and ulcer floors, fungus growth (microsporia)

Black.—Excessive skin pigment (Negro morbus addisonii, arsenical melanosis) oxidized keratin ("phaeoderma") in ichthyosis and blackheads, necrotic connective tissue (gangrene)

Macules which are caused *simultaneously* by hyperemia and hyperpigmentation become lighter under glass pressure, but they do not disappear completely Under the diascope their own color appears, as is the case in the color of a



Fig. 28.—Border of an erythematous spot under glass pressure



Fig. 29.—Border of pigmented macule (nevus) under glass pressure.

tuberculous cellular infiltrate of lupus vulgaris or the pigment of a nevus vasculopigmentosus. A discoloration surrounding a lesion is spoken of as a *halo* Thus we recognize pigmented (Fig 30) and depigmented (Fig 31) hemorrhagic, erythematous (Fig 32) and anemic (Figs. 33 and 34) halos. Occasionally the discoloration has a relationship to the hair follicles, which may appear darker (Fig. 35) or lighter (Fig 36) than the surrounding skin. Lividly erythematous halos surrounding the follicles are regularly seen in follicular keratoses (so-called lichen pilaris) and in chilblains (erythema pernio)

Erythema the anatomical basis of which is an increased filling of blood vessels (hyperemia) represents a transition between the lesions which are situated in the level of the skin and those which are raised (elevated) Erythema may be either macular or more or less urticarial in character The latter occurs when seepage of fluid from the blood vessels (edema) causes an elevation of the lesion. Blushing (erythema pudoris) is a good example of a purely macular erythema. However in some circumscribed erythemas (e.g., drug eruptions



Fig. 30.—Pigmented halo (macula anesthetica in leprosy)



Fig 31.—Depigmented halo (itiligo perinevica)

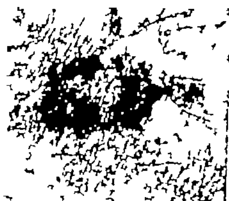


Fig 32.—Hyperemic (erythematous) halo (bullous mosquit bit)

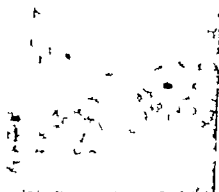


Fig 33.—Anemic halo (angioma senile)



Fig. 34.—Anemic and depigmented rea (psoriasis)



Fig 35.—Hyperpigmented follicles (nev pigmentosus)

and erythema exudativum multiforme) a slight elevation of the lesion can be noted (Fig 37) This can be termed an *urticarial erythema* or an *erythematous urtica*. If the erythema is inflammatory a cellular infiltrate may already exist, although clinically only a macule in the strict sense can be observed (syphilitic roseola, erythema exudativum multiforme) As the cellular infiltrate increases, the macule gradually becomes a papule (e.g the papular lesions of secondary syphilis) which means a raised lesion caused by an increase in cells (see below) Thus there exist gradual transitions not only from erythema to the wheal but also from erythema to the papule, the differences being quantitative in character

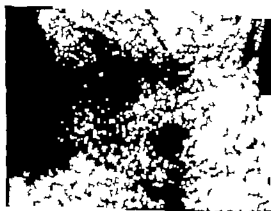


Fig. 36 —Hyperpigmentation respecting follicles (anterior aspect of neck)



Fig. 37 —Slightly elevated erythema (erythema exudativum multiforme)



FIG. 30.—Pigmented halo (macula anesthetica in leprosy)



FIG. 31.—Depigmented halo (vitiligo perinevica)

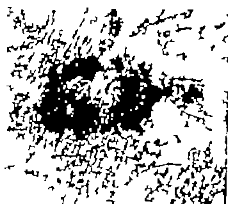


FIG. 32.—Hyperemic (erythematous) halo (bullous mosquito bite)

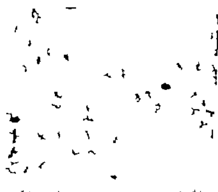


FIG. 33.—Anemic halo (angioma senile)



FIG. 34.—Anemic and depigmented area (psoriasis)



FIG. 35.—Hyperpigmented follicles (nevus pigmentosus)

rapid development and disappearance of the wheal, usually within a matter of hours. The size of the wheal may vary greatly. Only exceptionally is it so small that it can be confused with a papule (papuloid wheal. Fig. 39). For the most part, a wheal expands quickly and forms a coin-sized disk, but it may become much larger, exceeding the size of a hand. Frequently it enlarges peripherally. At the height of its development, the edges of the lesion are well defined, forming round or polycyclic figures (Fig. 40). The larger wheals are not higher in the center than at the edges but are frequently depressed because of central regression. However, if the subcutis participates in the edema, more rounded swellings (some larger than a hen's egg) with vague edges develop. The wheal feels firm and is non-pitting. However, if the edema involves areas with very loose cutis and subcutis, such as the eyelids, lips, and genitals, then prominent, spherical,



FIG. 39.—Urticarial wheal resembling papule (urtica papuloides) on the arm in urticaria.

somewhat translucent, frequently monstrous swellings (Fig. 41) develop. These are moderately soft, and the pit left by the pressure of a fingertip flattens slowly. These lesions are no longer called wheals but simply *circumscribed edemas*. Naturally they are often associated with wheals, since both represent the same fundamental pathological process. *Urtica mollis* is an apt dermatological term for this type of circumscribed cutaneous edema.

Wheals may have the color of the normal skin; they may be redder if the superficial blood vessels are dilated or paler if they are compressed by the accumulated extravasated fluid in the tissues. Accordingly, *urticaria hyperemica* or *erythematosa* (antiquated term "urticaria rubra") must be distinguished from *urticaria anemica* (obsolete term "urticaria porcellanea"). Like other lesions, the wheal may be surrounded by a hyperemic (erythematous) or anemic halo.

The wheal is the typical lesion of *urticaria*. In this disease, wheals develop spontaneously. In a minority of patients with urticaria, wheals can be provoked

The term *erythema* is used only if the redness is caused by reversible vasodilatation If the redness is produced by permanent widening lengthening and increase in number of the blood vessels the term *telangiectasis* or *telangiectasia* is used Such telangiectatic reddenings may also show transitions from the macule to elevated lesions. With increase in the caliber and number of the vessels involved the affected area is raised above the level of the surrounding skin and clinically it may give the impression of a papule or if large enough a tuber

Erythemas as well as macules may be of any *size* from tiny red points to very large areas even involving the entire body surface (scarlet fever) Erythematous eruptions consisting of numerous lentil to coin sized spots are not rare Such small-spotted disseminated erythemas are called *roseolas* (second



Fig 38.—Macular erythema (roseola) on forearm in secondary syphilis

ary syphilis, typhoid fever) The individual spots are located at points where an arteriole brings blood to the skin (Fig 38) Thus the roseola is in certain respects the negative picture of *cutis marmorata* (Fig 16 p 27) and vice versa *Erythema is the most common feature of eruptions. It is always present in inflammations and therefore precedes and/or accompanies many other lesions* (as an erythematous base or halo)

Raised lesions may develop by an accumulation of fluid in the skin (edema) or by an increase in the number and size of cells (cellular infiltrate) or cellular products (e.g connective tissue fibers) The edematous elevation is called an *urticarial lesion or wheal* whereas the solid lesion which consists of cells or cellular fibers is termed a *papule* The wheal is a circumscribed edema of the skin produced by the escape of blood plasma through the vessel wall It can be imitated by intradermal injection of liquids. Histopathologically the papillae in a wheal appear to be enlarged, the lymphatic spaces of the cutis widened and the epidermis swollen by serous infusion *Its pathogenesis explains the*

flesh (pilomotor reflex) This phase is quickly followed by an erythematous streak, the *erythema mechanicum*. This reaction is the vasomotor stimulation phenomenon or simple erythematous dermographism. In an erythematous skin the anemic phase may last a long time. If the mechanical stimulus is strong or if the reactivity is great, an anemic halo may develop (*white or anemic dermographism*) After about 2 minutes, in some persons, an edematous bulge rises in the middle of the streak and disappears after 15 or more minutes. This is *urticarial dermographism* The provoked friction wheal is frequently pale but has an erythematous halo and is sometimes surrounded by erythematous spots. It is only the more accentuated expression of a generally existing reactivity of the skin. Its significance is not fully understood. In the Middle Ages it was believed to be a sign of the devil and those afflicted with this disposition to cutaneous edema were burned alive. In modern times urticarial dermographism



FIG. 42.—Urticarial wheal with central vesicle in strophulus

has frequently been considered a stigma of nervous or psychic defects. According to our present knowledge factitial urticaria is without practical significance except in meningitis (Trousseau's phenomenon) The vasomotor stimulation phenomenon is increased in vasolabile persons and in patients suffering from toxic goiter and it is decreased in myxedema and in the senile skin.

The fluid in the lymphatic and interstitial spaces of the wheal has, as a rule no tendency to coalesce into a cavity and form a blister Yet in exceptional cases this blister formation may occur

It is known that sometimes the wheal of a mosquito bite develops into a marble-sized blister In strophulus, a small vesicle or papule forms in the center of an almond-shaped erythematous wheal. The intense itching causes this central vesicle soon to be excoriated (lesion of strophulus, Fig. 42) Occasionally an analogous transition from erythema to a blister can be observed. The erythemas of erythema exudativum (i.e. vesiculosum) multiforme often change to elevated urticarial disks in whose centers liquid collects in cavities. There is

artificially by pressure or friction This provocation of the lesions of a specific disease by non specific varieties of stimuli is called the *isomorphic effect* In America the term *Koebner s phenomenon* is often used while European dermatologists are inclined to reserve this eponym for psoriasis, where it was first observed This is essentially different from the *urticae mechanicae* which in some apparently healthy persons can be provoked and which may not itch.

Stroking the normal skin vigorously with the fingernail or with a blunt needle provokes a short anemic reaction which is sometimes accompanied by goose

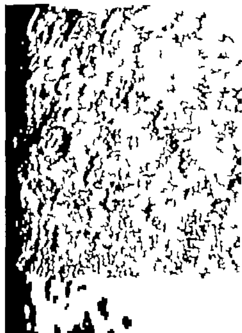


FIG. 40 —Wheals in urticaria



FIG. 41 —Edema of the skin 'urtica mollis'



Fig. 43.—Vesicles (herpes zoster)



Fig. 44.—Confluent vesicles with polycyclic edges (herpes simplex)



Fig. 45.—Confluent vesicles (eczema vesiculosum)



Fig. 46.—Vesicle with central depression (dermatitis herpetiformis Duhring)



Fig. 47.—Pocks, vesicles with central necrosis (vaccinia)

then a central blister surrounded by a wheal with an erythematous halo (Fig 225 p 131)

Vesicles are lesions which possess a macroscopic cavity filled with fluid (Fig 43) Since fluid in a cavity exerts equal pressure in all directions vesicles assume a spherical shape Larger vesicles or blisters may develop by coalescence of several small vesicles. In some cases they may show polycyclic (composed of several segments of a circle) borders (Fig 44) In other instances their development from several elements can be recognized by differences in color (Fig 45) Sometimes the roof of a vesicle is centrally depressed (Fig 46) If the depression is caused by central necrosis (variola vaccinia) the lesion is spoken of as a *pock*[1] (Fig 47) and the depression is called the *navel* of the pock (umbilicated lesion) Vesicles are usually tense, are elevated and have a taut and glossy surface. They are firm to the touch. If they are small and imbedded in thick and tough skin such as on the palms they may be neither raised nor palpable Yet they can *still* be recognized by their round shape and the glass-bead like reflections of their translucent contents (Fig 48) or by the little ring shaped collars of scales which remain after the vesicle has emptied (collarettes) Confirmation of the diagnosis of a vesicle is obtained by pricking the lesion with a needle and observing whether or not fluid escapes.

Vesicles have tops contents and floors. Their contents may consist of serum fibrin some cells and occasionally blood If the content dries a little round yellowish brown to dark red or brown crust forms at the center (Fig 49) If the top of the blister bursts or is scratched off the *floor* is exposed and appears as a small, round erosion. As soon as the exudate dries the erosion becomes covered with a small serous, or bloody crust which is not so perfectly round or smooth as the crust of the dried-out vesicle top It is characteristic of some diseases that the vesicle top bursts very early (impetigo) or is invariably scratched off the vesicles of other dermatoses are more stable dry without injury and exfoliate after the healing of the floor or are shed as a crust formed over the entire lesion In the pock necrosis of connective tissue causes deep destruction so that sharply bordered and depressed (varioliform) scars result (variola occasionally varicella *acne necroticans hydroa aestivale*) Mucosal vesicles usually lose their tops soon after they appear and are ordinarily seen as small round erosions whose floors are covered with a little pus or with a fibrinous (diphtheroid) exudate Mucosal vesicles or erosions of vesicular origin are called *aphthae*

Vesicles are located very superficially and almost exclusively involve various layers of the epidermis under the horny layer Vesicles have various modes of pathogenesis and are classified into types as *intercellular intracellular* and *necrobiotic* Intercellular edema (fluid between the cells) pushes the rete cells

1 The noun "pock" is not in general usage in America, though combinations like smallpox and the adjective "pockmarked" are well known among the laity

rapid growth (Fig 51) which can be checked by early incision and release of the pressure. This is done more effectively with fine scissors than by needle puncture which seals itself too easily Bullae can be provoked mechanically as can wheals, by prolonged rubbing (friction blisters on the hands from rowing or on the feet from unaccustomed marching) In patients with epidermolysis bullosa, minor rubbing or a slight bump is sufficient to provoke such blisters.

In these cases the great ease of mechanically producing blisters confirms the diagnosis. To accomplish this rub the fingernail in the opposite direction from that of scratching This is called *scratch-rubbing* By real scratching the epidermis would be injured. A suitable place for such scratch-rubbing is the skin over bony eminences, e g on the knuckles.

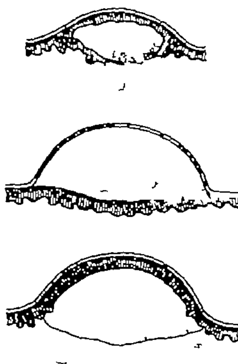


Fig 50 — Vesicle, subcorneal (intraepidermal) bulla, subepidermal bulla.

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come accustomed to insufficiently accurate observations, thus leading to unreliability This is also true of certain definitions of papule and pustule Some authors have proposed making the definition of the *papule* dependent on its later spontaneous involution. In this somewhat curious way the prognosis is made the premise of the diagnosis, instead of, as is more reasonably the case, the diagnosis the basis for the prognosis Furthermore, all small-sized solid elevations which do not regress spontaneously would be left unnamed, necessitating a new term. There have also been efforts to restrict the term *pustule* pathogenetically It has been proposed that only accumulations of fluid containing pus primarily should be called pustules, while all those vesicles and blisters which become purulent secondarily should receive a new name. This again would lead to confused diagnoses of lesions. Therefore, in defining lesions, the safe ground of clinical morphology should be the criterion.

apart forming a honeycomb or spongelike structure whose meshes finally rupture This process is called *spongiosis* and is typical of eczema. Intracellular edema arises within the cells. Thus unicellular 'vesicles develop which coalesce and form the clinical vesicle. An example of this process is the so-called *altération cavitaire* (French change characterized by cavities) in variola. In other cases the edema arises *between* the cells but the cells undergo processes of necrobiotic deterioration which depending on the resulting cell forms, are termed *ballooning or reticulating degeneration* (varicella herpes) Vesicles also develop simply by separation of epidermal layers Vesicles may be of pinhead size or larger but if a liquid filled cavity is larger than a pea it is no longer called a vesicle but a *bulla* (blister bleb) Blisters may be of hen's-egg size or larger However they differ from vesicles not only in size but also in their often deeper location and different mode of development Quite large blisters may

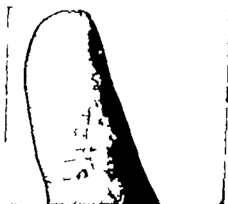


Fig. 48 —Skin-level vesicles (eczema vesiculosum)

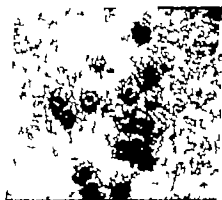


Fig. 49 —Drying vesicles (herpes zoster)

occasionally be situated directly under the horny layer More commonly they separate the deeper epidermal layers or are located between the epidermis and cutis lifting the epidermis in full thickness from its base.

Thus we distinguish subcorneal (keratolytic) intra-epidermal (acantholytic) and subepidermal (epidermolytic) blisters (Fig 50) Simple separation of the layers which is rarely found in vesicles is the most common pathogenesis of blisters. For this reason they are *in contrast to vesicles* usually unilocular There are however bullae of large size which are inflammatory in nature which originate from spongiosis (bullous eczemas) or which come about by intracellular (*altération cavitaire*) and interstitial edema with cellular disintegration (burn frostbite) The *size* of blisters varies greatly although by definition they must be at least as big as a pea [2] Some blisters have a remarkably

2 Some authors do not base their differentiation f vesicles from blisters simply on easily recognizable qualities *such as size* but rather on pathogenetic features such as cellular disintegration, on the one hand, and accumulation of fluid in a cutaneous interspace, on the other But th clinical diagnosis should, in my opinion, always be based on *morphological characteristics*. The pathogenesis frequently cannot be determined, which would then leave the diagnosis uncertain. The diagnostician would be

rapid growth (Fig 51) which can be checked by early incision and release of the pressure. This is done more effectively with fine scissors than by needle puncture which seals itself too easily Bullae can be provoked mechanically as can wheals, by prolonged rubbing (friction blisters on the hands from rowing or on the feet from unaccustomed marching) In patients with epidermolysis bullosa, minor rubbing or a slight bump is sufficient to provoke such blisters.

In these cases the great ease of mechanically producing blisters confirms the diagnosis. To accomplish this, rub the fingernail in the opposite direction from that of scratching This is called *scratch-rubbing* By real scratching the epidermis would be injured. A suitable place for such scratch-rubbing is the skin over bony eminences, e g on the knuckles.

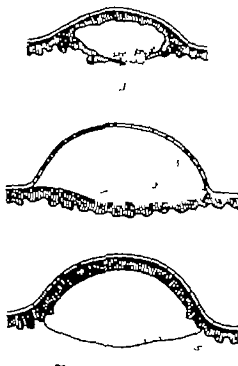


Fig 50 Vesicle, subcorneal (intraepidermal) bulla, subepidermal bulla

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come accustomed to insufficiently accurate observations, thus leading to unreliability This is also true of certain definitions of papule and pustule. Some authors have proposed making the definition of the *papule* dependent on its later spontaneous involution In this somewhat curious way the prognosis is made the premise of the diagnosis, instead of as is more reasonably the case the diagnosis the basis for the prognosis Furthermore, all small sized solid elevations hich do *not* regress spontaneously would be left unnamed, necessitating new term. There have also been efforts to restrict the term *pustule* pathogenetically It has been proposed that only accumulations of fluid containing pus primarily should be called pustules hile all those vesicles and blisters which become purulent secondarily should receive new name. This again ould lead to confused diagnoses of lesions. Therefore, in defining lesions, the safe ground of clinical morphology should be the criterion.

In epidermolytics after a few strokes of scratching the detachment of the epidermis can be felt by the presence of a shifting little epidermal fold. The potential cavity indicated by this fold fills up in a few minutes to form a blister which may continue to grow for several days (Fig 52) In pemphigus the findings are *sometimes similar* but the so-called Nikolsky sign is more typical This phenomenon elicited by a vigorous 'wiping' pressure of the thumb involves detachment and shifting of the uppermost layer *of the epidermis, leaving* a



Fig. 51 —Growing bulla (epidermolysis bullosa dystrophica)



Fig 52 —Bulla mechanically elicited by scratch rubbing in epidermolysis bullosa dystrophica

slightly eroded surface Blisters may also be caused chemically (cantharides) thermically (burns frostbites) and actinically (sunburn X ray) or they may form spontaneously i.e without any external cause Thus besides the inflammatory blisters caused by chemical toxic and infectious *agents mechanical* (epidermolysis) actinic (hydroa) and spontaneous (pemphigus) blisters can be distinguished.

Blisters, like vesicles have a round or sometimes oval *shape* (Fig 53) If they

are caused by the confluence of several blisters, their borders are more or less polycyclic (Fig 54) Very irregular jagged, and streaky outlines suggest extrinsic causation (friction, adhesive tape, cantharides meadow plants and in America, poison ivy) Fresh bullae are taut but soon become soft and wrinkled (Fig 55) If they grow very soft the fluid may collect at the lowest point, as in a bag (Fig 56) At the bottom of such a bag a cloudy accumulation of leukocytes may collect (hypopyon) Blisters may continue to dry more and more so that the loosely attached blister top when it finally tears, exposes an already slightly keratinized blister floor (Fig 57) If the blister top tears in an earlier stage the floor shows a glossy red oozing erosion (Fig 58) As is the case with vesicles the peripheral remnants of the blister top usually persist for some time as a ring of scales (collarette) It is from this coarse collar of scales that one can sometimes recognize that an erosion originated from a bulla (Fig 57 *B*) When the erosion heals, it leaves a residual erythema which may still show fine desquamation (Fig 57 *B*) sometimes leukoderma (Fig. 59) or melanoderma (Fig. 60) These residues are strikingly round and vanish slowly after weeks or months.

The contents of blisters like those of vesicles at first are clear yellowish or bluish or by admixture of blood reddish to black-red. However secondary infection with pyogenic cocci may cause such contents to become turbid from leukocytes. In this way the vesicle or the bulla may become a *pustule* (pus vesicle or pus blister) To emphasize the origin of a pustule from a bulla or to indicate its size, it can be termed a *bullous pustule* or a *purulent bulla*

There are lesions which develop primarily as pustules not only when the basic cause of the lesion is an infection (impetigo staphylogenous folliculitis, pustular trichophytosis) but also in some sterile purulent inflammations (e.g. mercury dermatitis) Regardless of whether primary or secondary the pustule is a lesion which contains a pus-filled cavity which is visible to the naked eye. Usually the pus cavity is located in the epidermis. If it lies so deep that the pus is invisible it is no longer called a pustule but an *abscess* (see below)

The *content of a pustule* is either a yellowish liquid which is more or less turbid from accumulated leukocytes or it is a creamy thick pus (Fig 61) Depending on the color of the pus the pustule may look white, yellow or greenish yellow If there is much necrosis one no longer speaks of a pustule but rather of *necrotic crusts* which, if they are small, resemble pustules (pustuloid crusts in acne necroticans and papulonecrotic tuberculids, Figs. 62 and 63) If the pustule does not perforate and if its top is not injured, the pus, like the serum in a vesicle will dry and form a brownish crust beneath this crust is found not merely an erosion, as in vesicles and blisters but frequently an ulceration. In this case the pustule will leave a scar after healing If there is no deep destruction of the floo of the pustule, the epidermis regenerates under the lesion from the periphery to the center Finally the whole original lesion is shed with the

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Fig 52 —Bulla, mechanically elicited by scratch rubbing (epidermolysis bullosa dystrophica

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A



B

Fig. 57.—Top (*A*) and epithelialized bottom (*B*) of blister ith still visible edge (epidermolysis bullosa dystrophica)



Fig. 58.—Erosion on the floor of bulla just torn open (epidermolysis bullosa dystrophica)



FIG 53.—Bullae (bullous mosquito bite)



FIG 54.—Confluent bullae (chemical burn by caustic ammonia)



FIG 55.—Wrinkled old bullae (impetigo bullosa)



FIG 56.—Flaccid pendulous bulla (pemphigus vulgaris)

crust. Since suppuration is regularly associated with a severe degree of inflammation, fresh pustules are surrounded by an erythematous halo which is less often found around vesicles and blisters and, when observed about these latter lesions, is much less marked.

In adults, pustules are usually related to hair follicles and in infants to the openings of sweat glands. If neither is the case they appear on the skin as small, lentil-sized, yellow disks with a red border (Fig 64) If the process of suppuration takes place in a hair follicle the pustule and the surrounding infiltrate are raised and resemble a cone (Fig 65 also note the conical shape of follicular papules Fig 78 p 59) Thus it is easy to distinguish *follicular and non-follicular pustules* A hair can often be detected in the center of a follicular pustule

The *acne papule* is a special type of follicular pustule In this lesion the hair follicle is closed by a horny plug Suppuration starts deep and is associated with a marked increase in inflammatory exudate and cells (Fig. 66) In the early stages, papule formation dominates the picture and only later does the pus which has been forming as a small abscess in the perifollicular cutis, appear as a pustule on the surface and finally drain out. Frequently the perforation does not take place in the center which is plugged, but rather somewhere at the side. Histopathologically the acne papule appears as a small perifollicular abscess, while clinically it is a combination of papule and pustule a *papulo-pustule*.

If a severe inflammatory process involves the follicle more deeply and causes necrosis and separation of a connective tissue sequestrum, we are dealing with a *boil* or *furuncle* The furuncle is a deep necrotizing type of folliculitis. When several adjoining furuncles coalesce to form a single focus the condition is called a *carbuncle* (Fig 67)

The general term *abscess* is used to describe a relatively large accumulation of pus in tissues and, with regard to the skin in particular to such an accumulation in the cutis and subcutis A sharp border exists between the pus cavity and the surrounding tissue. In order for a lesion to be an abscess, the pus should be located so deeply that it does not show on the surface. Because of its location, an abscess is scarcely raised above skin level and its borders are more easily palpated than seen (Fig. 68) On the surface the horny layer over an abscess may exfoliate, as may occur in any type of inflammation. Most abscesses develop from an inflammatory infiltrate by disintegration of cells and connective tissue, a process of colliquation necrosis the abscess wall is formed by the already affected, but not yet purulent tissue The colliquation, i.e. the transformation of a nodule into an abscess, manifests itself by central softening and fluctuation of the originally firm tissue. Sometimes it may be difficult or impossible to detect fluctuation if the abscess is very small or too tightly filled. As the abscess cavity enlarges, perforation and drainage of pus finally take place. Superficial abscesses open to the surface and form ulcers, while ones that are



Fig. 59.—Leukoderma after healed bulla (epidermolysis bullosa dystrophica)

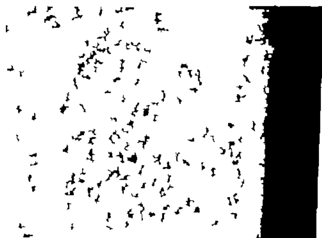


Fig. 60.—Melanoderma in the place f healed bulla (bullous mosquito bit)

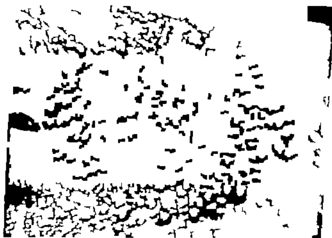


Fig. 61.—Pustules (trichophytosis pust losa) on the back of the hand

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Fig. 62.—Necrotic pustules (acne necroticans) on the temple.

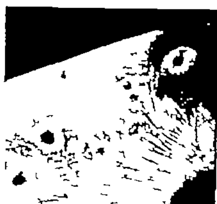


Fig. 63.—Necrotic pustules (papulo-necrotic tuberculid on the edge of the palm)



Fig. 64.—Non-follicular (streptogenic) pustule with erythematous halo (pyoderma)



Fig. 65.—Follicular (staphylogenic) pustule with cone-shaped infiltrate (furunculosis)

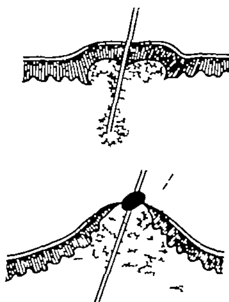


Fig. 66.—Folliculitis and acne pustules

deep give rise to sinuses (see below) An abscess which arises from a tertiary syphilitic infiltrate is called a *gumma* In France this rather unnecessary term is also used for tuberculous and other infectious cutaneous abscesses.

If accumulations of pus form in preformed spaces (e.g Bartholin's gland) and not simply in tissue they are called *pseudo-abscesses* a term which might more probably be replaced by *cavity abscesses* or in many cases by *cystic abscesses*

A *cyst* is an encapsulated cavity filled with fluid cells, and cell products. The cyst is never of inflammatory nature, though occasionally it may become secondarily inflamed. Its capsule is a connective tissue membrane which is lined



Fig. 67 —Carbuncle on buttock



Fig. 68 —Abscess (sporotrichosis)

with epithelium or endothelium since it develops as a rule from dilated and cutoff parts of glands glandular ducts, blood and lymph vessels, or layers of epidermis The contents of a cyst consist of products of the capsule such as serum, lymph sweat fat, sebum, epithelial cells horny platelets, or hairs. According to the nature and pressure of its contents, the cyst may be hard, doughy or even fluctuant In contrast to an abscess, symptoms of inflammation, especially redness and tenderness in the vicinity are always absent. Because of the tendency of their liquid or pulpy contents to spread equally in all directions or because of their regular growth, cysts are spherical or egg-shaped. They may be as small as a mustard seed (milium containing a horny pearl Fig 69) or a pea (hydrocystoma, containing sweat) or they may be as large as, or larger than, a goose egg (atheroma filled with epithelial cells and sebum)

If the skin covering a cyst is very much stretched the follicular openings are enlarged and form shallow pits (Fig 70) In contrast to true atheromas which originate from epidermal cells displaced into the subcutis, sebaceous cysts form when clogged hair follicles dam up the outflow of sebum These sebaceous cysts may show the obstructed follicle opening as a point or a small elevation (Fig 71) These lesions are referred to as follicular atheromas false atheromas, as well as sebaceous cysts. Even if the point cannot be readily seen it frequently can be demonstrated by the extrusion of a thin thread of thick sebaceous material if the cyst is squeezed Some cysts are located so superficially and



Fig 69 —Small horny cysts (milia)



Fig. 70 —Sebaceous cyst with extended follicular openings (atheroma verum of scalp)



Fig. 71.—Sebaceous cyst with visible obstructed duct (atheroma folliculare) on the forehead

have so thin a capsule that clinically they seem to be vesicles (miliaria crystallina, hydrocystoma lymphangioma)

The *papule* is a small elevation above skin level caused by an increase in number and size of cells and cell products rather than accumulation of liquid It has a solid center Exceptionally small papules may be imbedded so deeply in the skin that they protrude practically not at all and can only be palpated. Papules may be very flat on the palms and soles they are flattened by pressure (Fig 72) Papules may have clearly demarcated edges (Fig 73) they may be constricted at the base (sessile) or they may even be pedunculated and pendulous if the stem is long enough (Fig 74) They may be elongated (filiform papule Fig 75) and be with or without a horny tip If they are almost level with



Fig 72.—Flat papule (verruca palmaris)



Fig. 73.—Sessile papule (lentigo) constricted at base



Fig 74 —Pendulous papule (cutis pendulans) skin tag, anterior axillary fold.



Fig. 75 —Filiform papule (verruca filiformis)

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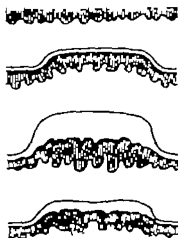


FIG 76 —Normal skin, urticarial wheal, epidermal papule (verruca) epidermido-cutaneous papule (lichen planus)

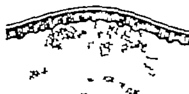


FIG 77 —Cutaneous papule (secondary syphilis)

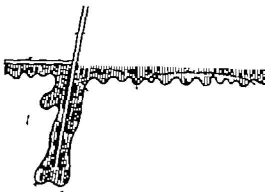


FIG 78 —Follicular and non follicular papule

the surface of the skin they may at first glance appear as a spot but on closer observation prove to be papular This is seen in the *lupus spot* which is translucent under glass pressure less resistant to probe pressure than the surrounding skin and therefore even if it is *not* raised is preferably called a *maculoid* (resembling a spot) a nodule rather than a macule

Other types of lesions may on superficial examination give the impression of being papules This sometimes occurs if a wheal remains very small and does not grow peripherally (Fig 39 p 41) Small erythemas may also appear to be slightly elevated (e.g the *papuloid* erythemas of erythema exudativum multiforme)

Papules may be so small that they are just perceptible The largest ones may reach the size of a lentil and are then called *lenticular* If they are still greater they are no longer called papules (see below) Papules may be red (inflammatory) or pale (anemic) hyperpigmented or depigmented or simply normal in color Some cellular infiltrates have a color of their own which becomes apparent only after the accompanying erythema is eliminated by glass pressure (lupus syphilis)

Papules are extremely multiform Their shape depends on the level of the underlying cellular infiltration. Therefore it is possible to draw conclusions as to the depth of the increased cellularity from the appearance of the papule. One can distinguish *superficial epidermal* and *deeper cutaneous papules* Superficial papules are plateau like with sharp edges Deep-seated ones are hemispherical with indistinct borders These differences can be well understood from histopathological considerations If the increase in cells is located mainly in the horny layer the keratohyaline layer and the rete of the epidermis as seen in verrucae then the border of the pathological process is clearly visible and appears sharply delineated (Fig 76) However if the accumulation of cells lies in the cutis as is seen in the papule of secondary syphilis then the entire thickness of the epidermis is lifted the center more than the periphery and the edges appear less distinct (Fig 77) Of course such a cutaneous cellular infiltrate tends to arrange itself horizontally under the epidermis, so that the small eminence which it causes forms a flat hemispheric prominence. If however the infiltrate is localized about a follicle it follows the follicle vertically downward into the depths of the skin These follicular infiltrates may also push themselves steeply above the surface. Therefore *follicular* papules are mostly cone-shaped and pointed (Fig 78) The follicular location of a disease focus can also be recognized from the pointed follicular opening or hair in the center (Fig 79) A dark plug or a light pointed even filiform scale in the center may take the place of the hair (keratosis follicularis acneiformis keratosis follicularis spinulosa lichen ruber acuminatus) If all follicles in a skin area appear to be papular the follicular character manifests itself by the strikingly regular arrangement of the lesions especially the equal distance between them (Fig 80)

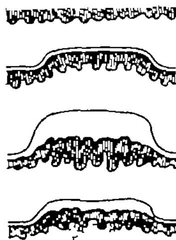


FIG. 76 — Normal skin, urticarial wheal, epidermal papule (verruca), epidermido-cutaneous papule (lichen planus)

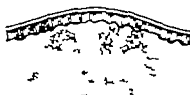


FIG. 77 — Cutaneous papule (secondary syphilis)

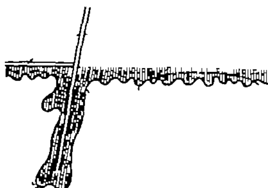


FIG. 78 — Follicular and non follicular papule

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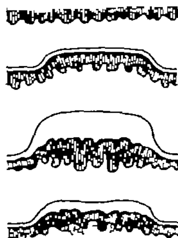


Fig 76—Normal skin, urticarial wheal, epidermal papule (verruca), epidermido-cutaneous papule (lichen planus)

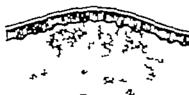


Fig 77—Cutaneous papule (secondary syphilis)

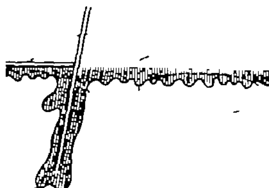


Fig 78—Follicular and non follicular papule

This picture resembles goose flesh (cutis anserina, Fig 81) in which follicles are forced to a more erect protruding position by contraction of their attached smooth muscles One could call these *physiological swellings* 'pseudo-papules. The different picture of a non follicular arrangement is shown by the quite irregular *positions* of the squamous papules in eczema (Fig 82) In spite of all these characteristics the decision as to whether an eruption is follicular or non-follicular may still be rather difficult in some cases. It may even be impossible without histologic examination

There are two types of papules which play an important part in practical diagnosis namely the lichenoid papule and the *eczematoid papule. The lichenoid* (lichen) *papule* is the characteristic primary lesion of lichen planus but is encountered *in a great variety* of papular rashes (lichenoid eczema lichenoid tuberculids lichenoid syphilids lichenoid trichophytids) It is a superficial epidermocutaneous papule The increase in cells takes place in the horny layer (hyperkeratosis) in the keratohyaline layer beneath (granulosis) in the other layers of the epidermis (acanthosis) and in the adjacent layers of the cutis (Fig 83) The cutaneous cellular infiltrate however fills at most only the papillae and a narrow subpapillary zone It then ends rather abruptly leaving the *deeper layers of the cutis almost free Infiltration of the cutis is accompanied by* exudation of fluid from the vessels This edema of the cutis is also very superficial involving mostly the papillae which swell and become mushroom shaped

Because of its histologic structure the lichen planus papule has the following clinical characteristics its surface is smooth and of a waxy glossiness since it consists mainly of a compact thickened horny layer This is possibly the reason why the lichen planus papule in spite of itching is rarely destroyed by scratching As the papules increase in size the granular layer which is thickened in some places, becomes visible through the horny layer as delicate spider web- *star or drop-shaped bluish or milky white figures* (Wickham s striae) These striae become more visible *after the horny layer is cleared with a droplet* of oil The shape of the lichen planus papule is *plateau like* hence *the term lichen planus* (Fig 84) From above it does not appear round but polygonal or mosaic like (Fig 85) because its borders follow the surface relief of the skin Quite frequently the lichen planus papule has small extensions which may make it star shaped. The characteristic flat and polygonal form is best observed in the smallest papules especially if tangential lighting makes the smooth surface appear shiny (Fig 86) Often in order to make the diagnosis it is necessary to look *for* and find such 'primary lesions. In contrast to the eczematoid papule the lichenoid papule has no tendency to evolve into advanced types of lesions such as blisters pustules, *and erosions*

The *eczematoid* (eczema) *papule* i e the primary lesion of banal superficial dermatitis is located a *little deeper than the lichenoid papule* The horny layer is not thickened but there usually is parakeratosis with absence of the kerato-



FIG. 79 —Follicular papules with follicle mouths and central hairs (tar acne)

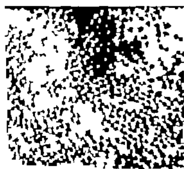


FIG. 80 —Regular arrangement of follicular lesions



FIG. 81 —Cutis anserina, goose flesh. All follicles are bulging.

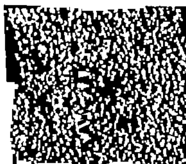


FIG. 82 —Non-follicular arrangement (scaly papules in eczema)

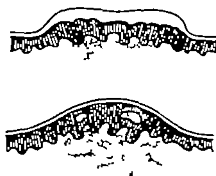


Fig. 83.—Lichenoid papule and eczematoid papule



Fig. 84.—Polygonal plateau-shaped lichenoid papule (lichen planus)



Fig. 85.—Polygonal plateau-shaped lichenoid papule (verrucae planae)



Fig. 86.—Tiny glossy lichenoid papules (parapsoriasis lichenoides atrophicans)

hyaline layer The increased cellularity in this type of papule is situated in the *deeper* layers of the epidermis (acanthosis) and also in the cutis. The infiltrate in the cutis does not end suddenly below the papillae but continues as a perivascular infiltrate into the depths (Fig 83) The accompanying edema is also much more extensive and is not confined to the papillae but spreads through the epidermis and cutis. In the epidermis the marked accumulation of fluid separates the cells forming small liquid-filled cavities (spongiosis Fig 83) which may coalesce into a larger cavity and convert the papule into a vesicle. Because of the succulence and the loosening of the epidermis and also the desquamation and thinning of the horny layer the eczematoid papule in contrast to the lichenoid papule is easily scratched open. Its center may become eroded or may be entirely shelled out by the scratching nail Finally it is replaced by a



Fig 87 —Excoriated eczematoid papules (eczema papulo-erosivum)

pinhead-sized round erosion with or without a crust (Fig 87) Thus the *clinical* differences between eczematoid and lichenoid papules can well be explained by the differences in their histologic structures.

The eczematoid papule is hemispheric and only exceptionally conical and pointed, if it happens to be follicular Usually it has an indistinct edge, so that the border line of the papule cannot be exactly defined (Fig. 88) The whole structure is less clear cut than in the lichenoid papule, and its surface is not glossy Its clinical course is marked by a tendency to develop into a vesicle because of spongiosis or into a pustule because of secondary infection.

In contrast to the stability of the lichenoid papule, the eczematoid papule runs a typical course the stages of which are erythema—papule—vesicle—(pustule)—erosion—crust—scale. In most eczemas one notices several stages existing simultaneously sometimes one can find the entire development of the lesion from erythema to scale recorded on the skin.

In papules of somewhat larger circumference, which also extend a little deeper a dome-shaped form becomes especially marked (papular secondary syphilis, larger verrucae vulgares, xanthomas) Some papules are depressed in



Fig 83 —Lichenoid papule and *eczematoid papule*



Fig. 84.—Polygonal plateau-shaped lichenoid papule (lichen planus)



Fig. 85 —Polygonal plateau shaped lichenoid papule (verrucae planae)



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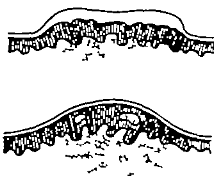


Fig 83.—Lichenoid papule and eczematoid papule



Fig. 84.—Polygonal plateau shaped lichenoid papule (lichen planus)



Fig 85.—Polygonal plateau-shaped lichenoid papule (verrucae planae)



Fig 86.—Tiny glossy lichenoid papules (*parapsoriasis lichenoides atrophicans*)

by the crowding together and confluence of papules. If lichenification has been produced by dense grouping of papules, it must like the lichenoid papule itself consist of increased amounts of cells either with or without inflammatory exudate. The thickening is easy to detect by raising a fold which feels thicker in the lichenified area than in a symmetric or otherwise comparable site. The very typical change of surface relief makes it possible to *see* the plaquelike infiltration of the skin. The finer furrows of the skin surface are smoothed out by the infiltrate and exudate which causes the few remaining ones to become deeper In other words, the surface relief becomes *coarsened* and reminiscent of shagreen leather (Fig 90) If the edge of the lichenified area is distinct, it can be noticed that the level of the changed skin is higher than that of the normal surrounding skin (Fig 92) If the transition to normal skin is a gradual one the difference between the levels can of course not be so clearly noticed and the coarsened skin relief gradually fades into the delicate hardly visible furrows of the normal skin (Fig 93) If the entire skin is lichenified, so that there are no borders at all one should compare it with the skin of a normal person (Fig. 94 *A* and *B*) The lichenification may be very fine (Fig 95) or coarse (Fig 90) The papules may become so large and the edges so rounded that they resemble the convolutions of the brain (hypertrophic lichenification Fig 96) If the thickening of the skin of the face causes deep cutlike furrows with bulging infiltrated skin in between, one speaks of *leontiasis* ("lion's skin" Fig 97) Because of the disappearance of the small furrows, lichenified skin is *smoother* and glossier (Fig 93) particularly if itching causes the patient to rub it constantly In other cases it is more dull and looks dusty from being covered with fine scales (Fig 98) Sometimes there are larger scales or even more coherent horny masses suggestive of warts (Fig 99) There may be point to pinhead-sized erosions on the altered skin, indicating the excoriation of individual papules or vesicles (Fig 100) The color of the lichenified skin area may be red, due to the inflammatory hyperemia, but it is more frequently pale and somewhat grayish. After a process of long duration, hyperpigmentation or rarely depigmentation develops. The surrounding skin may also be hyperpigmented.

Acanthosis and infiltration are not the only causes of a more pronounced accentuation and coarsening of the skin's surface relief It may also be brought about by other processes, e g a thickening of the horny layer (ichthyosis Fig. 101) Especially on the fingers, ichthyosis may resemble lichenification very closely and may be confused with it (Figs. 102 and 103) Also in nevi in cutis hyperelastica (rubber skin) and in old age wrinkles may produce a distinct pattern particularly in the face. The highest degree of coarsening of the skin's relief is encountered in cutis rhomboidalis nuchae (Fig. 104) In this condition it is produced by a fibrous coarsening of the degenerated connective tissue of the cutis.

The papule of prurigo is a variant of the eczematoid papule described pre

the center (Fig 89) or they have, in the center a small dull area or a larger area with irregular depressions (molluscum contagiosum)

Papules may be placed so close together that they no longer form separate prominences but instead a common plateau a papule overgrown in width. In this case the skin is infiltrated horizontally in width rather than in depth. This occurs frequently with lichenoid papules hence this condition of the skin has been called *lichenification* (also *lichenisation*) There may be small papules visible on the periphery (Fig 90) or groups of discrete papules can be found in the vicinity (Fig 91) which indicate that the skin change developed



Fig 88 —Hemispherical eczematoid papule on the forearm from home allergen

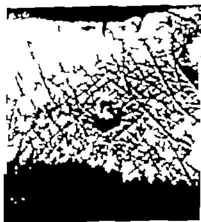


Fig. 89 —Papule with central depression (granuloma annulare of the thumb)

viously The *prurigo papule* is a more or less dome-shaped, rarely pointed (follicular) eminence with indistinct borders. It is not very striking and may easily be overlooked, especially because its color is frequently normal, though it may be reddish or brownish. Its presence can as a rule be recognized easily by the point to pinhead-sized, roundish erosions or bloody crusts which mark the location of excoriated papules (Fig 105) If a disseminated rash consists of



A



B

FIG 94—*A* universal lichenification in young girl (atopic eczema) *B* normal skin of the same region in young girl.

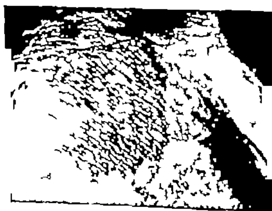


FIG 95.—Marked lichenification (eczema lichenification in the nape of the neck)

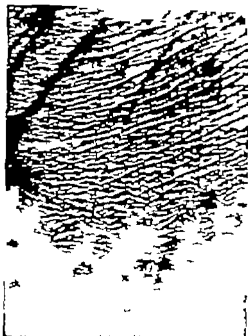


Fig. 90.—Lichenification (eczema lichenificatum)



Fig. 91.—Lichenification surrounded by groups of papules (eczema lichenificatum)



Fig. 92.—Lichenification with sharp edge (eczema lichenificatum)

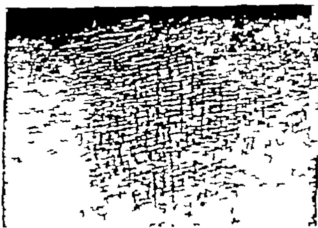




Fig. 99.—Verrucous lichenification (lichen planus)

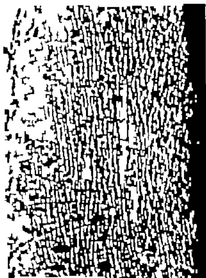


Fig. 100.—Lichenification with excoriated papules (coarse prurigo ... of the upper arm)

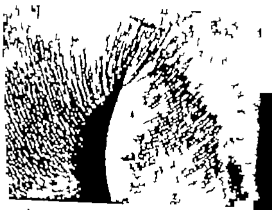


Fig. 101.—Coarsened relief of the skin from hyperkeratosis (erythrokeratodermia variabilis of the axillary field)



Fig 96.—Hypertrophic lichenification (eczema lichenificatum) on the medial aspect of the thigh



Fig. 97 —Leontiasis caused by lichenification (eczema lichenificatum)



Fig. 98 — quamous lichenification (eczema lichenificatum f the thigh)

excoriated papules resembling prurigo papules it is called *pruriginous* Prurig inous, therefore means 'papulo-erosive and this expression is usually used to designate eruptions of a kind which, in their morphology resemble prurigo Hebra and not the circumscribed lichenification of lichen simplex Vidal. If the prurigo papule is not eroded its surface like that of the papule of chronic eczema, is sometimes smooth and almost glossy but is more commonly covered with fine scales and dull. These clinical characteristics give clear indication that the histologic structure of the prurigo papule is the same as that of the eczema toid papule except for the more marked acanthosis and the tendency to lack vesiculation.

Eczematoid as well as prurigo papules may by apposition of groups of closely packed foci of infiltration, grow into larger papules which may then



Fig. 105 —Prurigo papule partly excoriated chronic papular eczem)

form larger than pea-sized hemispheric prominences This kind of lesion may have a smooth r excoriated surface Its relief may be coarsened but more fre quently it is dull from scale formation or it is pitted and even verrucous These large groups of papules which sometimes are closely grouped together and some times spaced more widely apart are called *nodular or obtuse papules* (Fig 106) (blunt papules, giant papules) They can be considered as circumscribed large-*papula areas l lichenification* They are either pale skin-colored, or pig mented and horny They have the disagreeable property of being very resistant to treatment and peculiarly much more resistant than lichenifications of larger size

The terminology of those solid eminences on the skin which are too big to be called papules (larger than a lentil pea or almond) is no longer so clear as for the lesions which have so far been discussed. Increases in cells and substances which have a fairly large size are by customary usage, called different names in different diseases I or deep-seated, well-defined papules up to almond size the



Fig 102.—Lichenification of the fingers (eczema lichenificato squamosum)



Fig 103.—Coarsened relief of the skin of the fingers from hyperkeratosis (ichthyosis congenita partly healed)



Fig. 104.—Enormously coarsened relief in cutis rhomboidea

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term *nodule* is used. The nodule is simply a subcutaneous or cutaneous-subcutaneous papule with well-defined borders A more voluminous papule in some diseases, is termed a *tuber* in others a *node* and in one case namely in the nodular hypertrophy of sebaceous glands and connective tissue of the nose one speaks of a *phyma* (bulb) It seems best to reserve the expression *tuber* for more superficial and the term *node* for deeper lesions of hazel nut and larger size. Consequently *tubers* are epidermo-cutaneous to cutaneous-subcutaneous nodes which either are raised only a little above the skin (disk tumor Fig 107) or are relatively highly elevated (ball tumor Fig 108) Some tubers may even be constricted at the base (Fig 109) In contrast to the raised varieties of large



Fig. 106.—Blunt (obtuse) papule (lichen obtusus sive nodularis)

solid lesions *nodes* are mainly or entirely subcutaneous (Fig 110) and may even involve muscle or bone. Because of their deep situation such masses have a very flat dome (Fig 111) if they rise above skin level Tubers may be flat representing in a way laterally overgrown papules or they may be hemispheric or knobby prominences Thus one may distinguish *disk tubers* and *ball tubers* The disk tuber should be a little deeper than an ordinary nodule because otherwise it would only be lichenification

The anatomic bases of these node formations may be extensive inflammatory cellular infiltrates (tuberous syphilis erythema nodosum) cellular neoplasms or tissue hypertrophies They frequently have a destructive character and break down with suppuration Tubers may become deep punched-out ulcers with scalloped edges (tubero-ulcerative syphilis) Nodes may form analogous nodular ulcerative processes (e.g erythema induratum Bazin) or still more



Fig. 107.—Discoid tuber with depressed center (tertiary syphilis)



Fig. 108.—Spherical tuber (xanthoma tuberosum of the elbow)



Fig. 109.—Sessile tuber constricted at the base (lupus tumidus)

commonly, abscesses and sinuses Such nodes with a marked tendency to form sharply marginated ulcerations abscesses and if deep-seated sinuses are frequently also called *gummas* a term which in the French literature is used for all abscess-forming nodes of infectious nature though it is elsewhere generally used only for syphilitic lesions of this kind (see p 55)



Fig 110 —Node, bulging (Von Recklinghausen s disease)



Fig 111 —Node flat (erythema nodosum)

It is certainly permissible to call all large masses of inflammatory tissue *infiltrates* The term *tumor* is a very general one covering all *other* increases in cells and tissue including those whose nature has not yet been determined infiltrates (tubers and nodes) tissue hypertrophies (elephantiasis Fig 112 rhinophyma) and even edematous swellings as long as they are of large size. If they are small the word *tumor* is reserved for true neoplasms. In this case *tumor* designates not only a lesion but also a diagnosis of a definite category

of disease namely neoplastic. This is usually expressed by the ending *-oma* Wherever the word *tumor* is encountered one always has to consider whether the lesion or the disease is meant.

In addition to these small and large prominences—ranging from the papule to the tumor—there are also eruptions which consist of many small, closely packed, sometimes round, sometimes pointed or threadlike, projections. These formations are called *vegetations* Vegetations may be of fine or coarse caliber they may appear in small rounded aggregations looking like raspberries (frambesiform) or they may form beds covering areas of almost any size. Vegetations may be found below the level of the skin in wounds and ulcers (granulations)



Fig. 112—Swelling (elephantiasis accompanying lupus vulgaris)

they may simply sit on the skin (ichthyosis vegetans) or they may also occur on top of other prominences, such as nodules and tumors. The vegetations in ulcers, which are called *granulations* because they are granulated, are glossy and bright red (Fig. 113) They represent an early stage of scar tissue and are rich in blood vessels and cells which promote secondary healing Subsequently the final scar tissue develops, which is poor in blood vessels and cells. If vegetations become excessive, they are referred to as *hypertrophic granulations* or *proud flesh* (caro luxurians) Vegetations sitting on non-ulcerated skin, even on papules and tumors, including pedunculated ones, often appear bright red, glossy and moist These are naked or *erosive vegetations* They may be bulging and smooth (Fig. 114) or consist of round or pointed papules (Fig. 115) If they have developed on the floor of a blister the remnant of the blister top is usually still visible as a coarse collar of scales (collarette Fig 114) The surface of

vegetations may be ulcerated (ulcus molle elevatum) The entire bed of vegetations may be constricted at the base or may be situated on a stem reminiscent of a raspberry or a cauliflower or if the surface is smooth of a pea or a marble (granuloma telangiectaticum Fig 116) If the vegetations are covered with intact epithelium they are of course dry and more or less skin-colored These are called *papillomatous* or *papulous* vegetations Papulous vegetations may be plateau like, flat and more or less round (lichenoid) (Fig 117) or they may tend to be pointed (Fig 118) They also may sit on a stem and thus resemble a raspberry or mulberry (Latin *morula*) (Fig 119) If the horny layer of the vegetations is thickened they feel hard and have a yellow gray or even black color These are called *verrucous or keratotic* vegetations (verrucosities, Fig 120) In mild cases the surface looks pitted (Fig 121) If the keratosis is thicker the elevations become granular (Fig 122) or even pointed like spines or filaments (Fig 123) Ordinary papular vegetations later still may convert into keratotic ones (Fig 124) Sometimes vegetating excrescences are called *papillary* This expression is likely to lead to the idea that such excrescences are a visible picture of the elongated papillae of the cutis In reality however though the papillae actually are lengthened each of the vegetations corresponds to *several* papillae which have merged on the same connective tissue and vascular shoot Therefore it seems better to avoid the word *papillary*

The lesions of skin diseases may also bring about a *depression* of the skin level This is most frequently the case in all surface defects. Such losses of substance may extend to different depths If one only lightly scratches the skin surface one will remove only the horny layer i e scales The result is a dry streak surrounded by horny layer debris (Fig 125) If the scratching nail penetrates deeper into the stratum acanthoticum (stratum malpighii Fig 126) of the living epidermis the track of the scratch will be moist with clear serum oozing forth If the injury reaches still deeper the papillae are torn off Thus a punctate or if a larger area is involved sievelike hemorrhage results. These superficial lesions are called *erosions* (Fig 127) If the injury penetrates into the connective tissue proper the bleeding is more uniform and copious. Then we speak of a *wound* (Latin *vulnus*)

Erosions are not caused exclusively by trauma, but they may develop by detachment of the uppermost epidermal layers because of maceration (eczemas between the toes and under the mammae balanitis erosiva and inflammatory processes (syphilitic primary lesion Fig 128) Most frequently erosions originate in the epidermis or between epidermis and cutis from a vesicle or bulla which has lost its top (Fig 58 p 51) The vesicular or bullous genesis of an erosion can be diagnosed by its circular outline and the remnant of the blister top (collarette coronella or corolla) which is often still present at the edge However such an epithelial collar around erosions does not necessarily tell the



Fig 113 —Granulations on the floor of an ulcer



Fig 114 —Erosive vegetations with evenly b surface and collarette (bullous bromide eruption)



Fig 115 Erosive vegetations with pointed projections (condylomata acuminata



Fig 116 —Erosive and hypertrophic vegetation (granuloma telangiectaticum)



Fig 117 —Papular vegetations with rounded projections (elephantiasis of the leg)



Fig 118 —Papular vegetations with projections (evus papillomatosus)

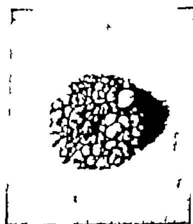


Fig 119 —P pular vegetations (lentigo morula)



Fig. 120 —Keratotic vegetations, coarsel grained (evu ichthyosiformis on the up er rm)



Fig. 121.—Pitted, keratotic vegetations (verrucae vulgares in an early stage on finger)



Fig. 122.—Granular keratotic vegetations (dermatitis from gold)



Fig. 123.—Spiny keratotic vegetations (verruca vulgaris hystriciformis)



Fig. 124.—Keratosis following papular vegetations (epidermolysis bullosa dystrophica vegetans)

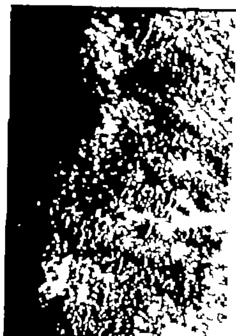


Fig 125.—Squamous linear scratch marks on normal skin



Fig 126.—Excoriated scratch marks on normal skin



Fig. 127.—Defects of varying depth (1) desquamation (2) erosion, (3) excoriation (4) wound

tale of its development from a blister as the examples of balanitis erosiva or erosio interdigitalis blastomycetica show In these conditions the epithelial collar is caused by maceration. Erosions following vesicles of mucosal surfaces (aphthae) often lack the collarette but retain the round or oval outline

It is characteristic of some postbullous erosions to form vegetations instead of healing smoothly (pemphigus vegetans vegetating bullous impetigo bullous bromoderma, Fig 114) In other instances healing of the blister floor is accompanied by the formation of tiny epithelial cysts (epidermolysis bullosa dystrophica traumatic scars)

Erosions should not be confused with certain deep desquamations which in inflammatory diseases like lupus erythematosus, may also look dark red and glossy These are dry which can easily be demonstrated by touching them and are called *pseudo-erosive desquamations* (Fig 129)

Although scratching tearing or abrasions may cause *excoriations* in normal skin, they do this much more readily when the papillae of the cutis are elongated so that their tips can be easily torn off (Fig 130) This situation is most pronounced in psoriasis. Therefore, punctate sievelike bleeding on vigorous scratching is regarded as a diagnostic sign of psoriasis and this type of bleeding is called *psoriasiform bleeding*

After healing erosions and excoriations frequently leave hyperpigmentations or depigmentations (melanodermas or leukodermas) which may persist for some time (Figs 59 and 60 p 52)

Fissures or *rhagades* linear cracks caused by excessive tension or diminished elasticity in the skin may be classified with the erosions and excoriations. They are mostly found where the elasticity of the skin suffers the greatest strain i e in the natural folds of movement over the joints and on the transitional areas between skin and mucous membranes (angle of the mouth anus) They always run crosswise to the direction in which the skin is stretched and may be very superficial extending no deeper than the horny layer which may simply burst apart in the face of severe swelling of the underlying tissues (Fig. 131) I all these cracks *fissures of the horny layer fissurae squamosae* (Fig 196 p 113) However cracks may reach into the living epidermis and even into the cutis causing oozing bleeding, and smarting These are the fissures proper *fissurae excoriatoae* (Fig 132)

According t their described mode of development fissures represent only a loss f continuity a crack of the skin as a rule without any loss of substance. Often they are located on an infiltrated (psoriasis eczema Fig 132) or papular and elevated base (syphilitic papules in the angles of the mouth) An inflammatory nfiltrate is the most frequent cause of the loss of elasticity which favors the tearing of the skin. In other cases the cause may be found in excessive tension n the less elastic horny layer e.g over acutely inflamed and swollen skin r on shrunken scars. It should be noted that the stratum corneum is the



FIG 128 —Erosion (syphilitic chancre)



FIG 129 —Desquamation resembling erosion (lupus erythematosus)

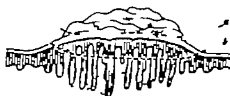


FIG. 130.—Psoriasis lesion a zone of brittle and dry desquamation b zon of bleeding points

least expandable layer of the skin and is chiefly able to extend its surface only by straightening the skin relief furrows. If these furrows are absent, as is often the case on scars, the horny layer is bound to tear if it is stretched to any extent. In the depths of excoriative fissures the rete malpighii or the uppermost part of the cutis lies bare oozing or bleeding like all other erosions and excoriations. Rarely the cracks become deep enough to leave thin linear and furrowed



FIG 131 — Fissures, squamous (erysipeloid)



FIG. 132 — Fissures, excoriative (eczema keratoticum)

scars. This type of scarring around the mouth is often sufficiently characteristic of congenital syphilis to make a diagnosis possible (Parrot's furrows in congenital syphilis). Of course fissures may become secondarily infected and ulcerated (ulcerated fissures). It is of great importance that chronic fissures can be the portals of entry for serious infections with subsequent lymphangitides and recurring attacks of erysipelas.



FIG 128.—Erosion (syphilitic chancre)



FIG 129.—Desquamation resembling erosion (lupus erythematosus)

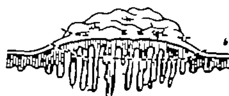


FIG 130.—Psoriasis lesion   zone of brittle and dry desquamation *b* zone of bleeding point

abscesses The simultaneous influence of mechanical injury and irritant secretions and salves may provoke the symptoms of a papulovesicular eczema (*scratch eczema*) thus adding *eczematization* to impetiginization and creating a very heterogeneous syndrome which may camouflage the original skin disease (pediculosis, scabies, eczema) In the case of a longer lasting ailment with constantly repeated rubbing and excoriation as, for instance in untreated pediculosis vestimentorum, the cumulative effect of the irritation causes chronic infiltration, hyperpigmentation and hyperplasia of epidermis and cutis The skin of the involved areas takes on a dirty brown color interspersed with

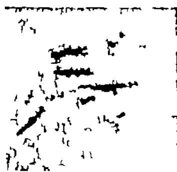


Fig 133 —Linear excoriations from scratching on normal skin (eczema)



Fig 134 —Follicular excoriations from rubbing on normal skin, regular arrangement.



Fig. 135 —Round excoriations from scratched papules and crusts (eczema papulo-vesiculosum) Irregular arrangement.



Fig 136 —Leukoderma from scratching and excoriations (eczema papulosum disseminatum on the shoulder)

Erosions and excoriations appear most frequently and in largest numbers as the result of scratching (*scratch marks*) Itching however does not always lead to scratch marks In this respect there exist two entirely different types of itching (1) itching which subsides on mere rubbing pressing or kneading and (2) itching which induces scratching with the power of a categorical imperative (Hebra) *Rub itching* is characteristic of urticaria and some lichenifications, while *scratch itching* occurs in scabies pediculosis vestimentorum strophulus, and most of the chronic papular eczemas The shapes of the scratch marks are often typical depending on the nature of the itching lesions and are therefore of diagnostic significance On morphologically normal skin scratch streaks form and if they go deep enough bleed and become crusty (Fig 133) Rubbing tends to excoriate the follicles leaving small round crusts which like all follicular eruptions, are arranged at strikingly equal distances from one another (Fig 134) If papules and vesicles are destroyed by scratching as, for instance in strophulus and in chronic papular eczemas punctate and small circular scratch marks appear These marks are not so regularly arranged as are the follicular ones (Fig 135) The scratches evoked by hives are frequently oblong *with pointed ends and are wider in the middle where the scratching* nail can penetrate most deeply into the edematous tissue If the scratching is very violent, one mostly sees long parallel or sometimes crossing groups of scratch marks Some of them may be only scaly others may be covered with thin bloody scabs but still their parallel arrangement indicates simultaneous scratching with several fingers. Frequently scratch marks leave leukodermatic (Fig 136) melanodermatic or leukomelanodermatic spots or even irregular small scars

Wherever scratches or destroyed vesicles or blisters *sever the continuity of the epidermis* a portal of entry for secondary infection by pyogenic cocci exists. Therefore all itching skin eruptions which lead to scratching are likely to be complicated by pustular and crusty pyodermas. This process of secondary suppuration is called *impetiginization* a rather unwieldy term which might better be replaced by *pyonization* (pus infection from Greek *pyon* 'pus) because it does include not only the impetiginous (i e the superficially crusty changes) but also every possible type of secondary pyogenic infection including the deep (ecthymatous) and follicular (furuncular) involvements If pyogenic infection has set in the small wounds which were covered with little thin dark-red, bloody scabs become covered with thick moist turbid brown to greenish purulent crusts surrounded by inflammatory red halos These lesions may show a tendency to spread independently in the local vicinity They may cover an ulceration of the skin containing a copious concentration of pus. There may also develop multiple lesions of poritis and folliculitis boils, and other forms of cutaneous abscesses The continued scratching spreads the pyogenic infection giving rise to primary impetigo blisters and crusts folliculitides and

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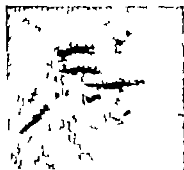


FIG. 133.—Linear excoriations from scratching on normal skin (eczema).

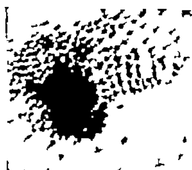


FIG. 134.—Follicular excoriations from rubbing on normal skin, regular arrangement.

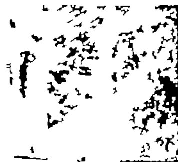


FIG. 135.—Round excoriations from scratched papules and vesicles (eczema papulo-vesiculosum) irregular arrangement.



FIG. 136.—Leukoderma from scratching and excoriations (eczema papulosum disseminatum on the shoulder).

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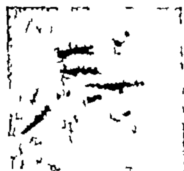


Fig 133 —Linear excoriations from scratching on normal skin (eczemas)



Fig 134 —Follicular excoriations from rubbing on normal skin, regular arrangement.



Fig 135 —Round excoriations from scratched papules and vesicles (eczema papulo excoriatum) irregular arrangement.



Fig 136 —Leukoderma from scratching and excoriations (eczema papulosum disseminatum on the shoulder)

numerous light, small scars This skin is rough desquamates easily and becomes thickened and hardly pliable (cutis vagantium vagabond s disease)

A *wound* (Latin *vulnus*) which is not so small as a scratch usually does not heal by primary intent. In larger injuries, as a rule secondary pyogenic infection of the wound floor sets in, followed by granulations which slowly fill in and heal the defect. Such a purulent and granulating wound is sometimes called an *ulcer* (Latin *ulcus*) especially if there is a distinct breaking-down of the edges and of the floor The term is also frequently used for necrotic defects due to burns in the stage of healing with granulation and pus secretion However only a suppurating defect which extends into the cutis should be called an *ulcer* in the strict sense if it originated from the breakdown of pathologically changed tissue. In this case one is likely to find in the border or the floor of the ulcer remnants of the pathologic change e.g the bulging neoplastic edge of a carcinoma or the inflammatory infiltrate of a syphilitic gumma The tissue-breakdown which leads to an ulcer is most commonly caused by liquefaction (colliquative necrosis) of an inflammatory infiltrate or a tumor The majority of skin ulcers do not start on the surface but rather from below The deeper the starting point of the liquefaction lies the deeper will be the ulcer and the steeper its edges If the ulcer is so deeply situated that it is invisible and connected with the surface merely by a ductlike opening it is called a *sinus* (fistula) Thus a sinus is the excretory duct of a deep-seated ulcer It originates from an abscess which perforates to the surface.

Ulcers may show very different appearances Their *color* may be blood red or yellow to brownish gray or black depending on the vascularization and the nature of the broken-down tissue. The *floor* may be clean meaning barely covered with a little thin pus or may look dirty from copious thick pus, pseudomembranes (diphtheria) detritus, and gelatinous accumulations and necrotic bits of tissue which may even include sequesters of muscle and bone The *edges* may be flat and smooth declining gradually from the normal level to that of the affected skin (centrally ulcerated primary lesion of syphilis) or the edge may be steep punched out or overhanging and undermined (tuberculosis injury by subcutaneous injection Fig 137) or sharply punched out and jagged (ulcus molle) If the broken-down tissue originated from a single focus of liquefaction the shape of the ulcer will be roundish (Fig 137) if it started from several foci the edges are likely to be polycyclic and of unequal depth. If the ulcerative process tends to scar on one side and to progress peripherally on the other side serpiginous and kidney shaped ulcers (tertiary syphilis) will result The *size* of ulcers varies widely While some types never grow to larger dimensions others tend to eat away relentlessly laterally or downward (*phagedenic ulcers*) Progressive growth may also be produced by the growth of a centrally ulcerated tumor whose enlargement also makes the ulcer grow

An ulcer is never found in completely normal skin. At least the immediately surrounding area is inflamed, red swollen, and infiltrated. This marginal infiltration may appear as a narrow ridge (epithelioma) or as a wide firm elevation (tertiary syphilis, epithelioma, Fig 138) sometimes containing horn pearls (epithelioma) or it may be piled up as a voluminous tumor mass (epithelioma gigantocellulare Fig. 139)

Beyond this marginal area the surrounding skin may also be infiltrated or of a boardlike sclerotic (Greek *scleros* "hard") consistency as in ulcus cruris It may also be hyperpigmented, depigmented, or eczematized. Sometimes exudate pus, blood, and debris have dried and formed a solid crust which has to be removed in order to reveal that there is an ulcer underneath. In exceptional cases the floor of an ulcer may vegetate to such a degree that it rises above the level of the surrounding skin (ulcus elevatum) giving the impression that there is no defect but rather an increase in tissue substance in spite of the absence of the upper layers of the skin (proud flesh ulcus molle elevatum neoplasma)

Ulcers reach the subpapillary layer of the cutis, and therefore they heal with a scar The *scar* (cicatrix) is only incompletely regenerated not fully intact tissue. Scarred cutis is poor in nuclei and blood vessels. The elastic fibers are either absent or fail to be arranged in an orderly network, and other connective tissue fibers are interwoven like felt or run parallel to the surface. The normal distribution of blood vessels also does not return in this scarred fibrous tissue the papillae of the cutis are left unregenerated and the epidermis, which had been thinned down to a few layers of cells, therefore covers the scarred surface without rete ridges. Furthermore cutaneous scars often lack the follicles with their hairs, sebaceous glands, and muscle fibers. Sometimes the remnants of follicles can be detected as flat distended depressions.

In summary scars consist of rarefied, thinned epidermis over cutis without papillae, follicles or glands. They are poor in blood vessels and elastic fibers. Scarred skin has no surface relief which would normally be formed by follicle openings as well as the furrows which correspond to the papillae and rete ridges. Hence scars are smooth and glossy (Fig. 140)

In contrast to spots of leukoderma, where surface relief and follicular pore pattern is normal (Fig 141) scars are completely devoid of follicle openings (Fig 142) or when present, these are distended into flat, hairless pits (Fig 143 *A*) If one tries to pull together the skin of some scars between thumb and index finger fine crisp glossy wrinkles like those of crushed cigarette paper become apparent, because the epidermis is thin and dry (Fig. 143 *B*) At times these wrinkles may be particularly fine (Fig 144) In some cases the epidermis is so tightly attached to the underlying fibrous connective tissue that wrinkles can hardly develop. The scar character of such a lesion can be recognized from the fact that relief less epidermis can no longer be wrinkled over its dense immov



Fig. 137.—Ulcer of the thigh (injury from subcutaneous injection of caustic)



Fig. 138.—Ulcer with rolled border (squamous cell epithelioma)



Fig. 139.—Ulcer with tumor like edge (squamous cell epithelioma)



Fig. 140.—Smooth trophic scar after treatment of lupus of the lower leg with the diathermy loop



Fig. 141.—Leukoderma with follicular pattern (after scratch marks in strophulus)



Fig. 142.—Atrophic scar partly smooth, partly with widened follicular openings (after injury)



A



B

Fig. 143.—B, atrophic wrinkling on pinching the skin together (scar after strong tuberculin reaction with Ponndorf method). A, without pinching.



Fig. 144.—Atrophic wrinkling, very intense (acrodermatitis atrophicans)

able underlying tissue In this case a combination of epidermal atrophy and cutaneous fibrosis or even fibromatosis exists, which is called a *sclerotic* or fibrous *scar*

If the scarring process involves only the follicles, follicular pits result which are of varying size some being quite large deep and funnel-shaped These are called *follicular scars* (Fig 145) They should not be confused with enlarged follicular openings which in some persons occur on the nose cheeks, and forehead and have a much more uniform appearance (Fig 146) If the follicular scar pits are so dense that they merge, the skin takes on a worm-eaten (vermicular) appearance (Fig 147) Very large round pits with sharply outlined steep edges may follow these vesicles or pustules which result from necrosis of the underlying connective tissue (variola varicella acne necroticans, Fig 148) They are especially characteristic of smallpox (variola) which explains the term *varioliform scars* In general they are not follicular but they may also be seen after follicular inflammations (acne vulgaris, Fig 149) Remnants of destroyed follicles and displaced epithelium may give rise to scar comedones, double comedones giant comedones, or milium like horny cysts which follow bullae. After an ulcerative breakdown the skin edges may join together in irregular fashion forming coarse fascicles or delicate skin bridges.

At first, the color of scars is red but in the course of months it grows paler Finally the color may become as white as marble because the connective tissue, which has lost most of its blood vessels, reveals its own color through the thinned epidermis. Sometimes this connective tissue by degeneration takes on a yellow hue The pale color of anemic connective tissue is especially conspicuous because in general the thinned overlying epidermis no longer forms pigment Nevertheless irregularly distributed hyperpigmented spots and halos are not too rarely found in scars and in their immediate vicinity Furthermore since lengthened and widened blood vessels (telangiectases Fig 150) frequently develop in cutis which otherwise has very few blood vessels, a characteristic dappled picture may develop The triple-colored appearance is composed of the brown of hyperpigmentation the white of depigmentation and anemia and the red of telangiectases. This combination which is more than a mere color change and which always indicates a certain degree of atrophy is called *poikiloderma* (from *poikilos* Greek for "varied irregular") or *variegated atrophy* It may occur as an independent disease entity but it is typical of X ray and radium scars (Fig 151) and also of xeroderma pigmentosum in which because of a certain hereditary sensitivity ordinary sunlight causes the same effects in the skin (variegated atrophy later carcinoma formation) as those which in normal people are produced only by X ray exposure and radium exposure Thus xeroderma pigmentosum represents what might be called a *poikiloderma solare* as opposed to X ray poikiloderma

Mutilations are caused by the destruction of deeper structures They may be



Fig. 145.—Follicular pitted scars (acne vulgaris).



Fig. 146.—Enlarged follicle mouths.

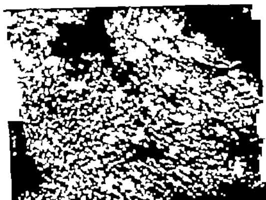


Fig. 147.—Wormeaten (vermicular) appearance of scars in acne vulgaris.



Fig. 148.—Varioliform scars (acne necrotica).



Fig. 149.—Varioliform scars (acne vulgaris of the back).

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Mutilations are caused by the destruction of deeper structures They may be

In a certain way scars can be considered as types of lesions in a category between those lesions which are always raised *above* and those which are always sunk *below* the level of the skin If the cutaneous connective tissue is thinned a depression no matter how minimal will result This is the *atrophic scar* (Fig 140 p 88) Frequently at other times in scar formation a hypertrophic reaction I connective tissue ensues, causing the scar to bulge like a tumor This results in a *hypertrophic scar* (Fig 154) The hypertrophic scar is a fibroma (more properly a *collagoma* because only the collagenous fibers are thickened) which is poor in blood vessels nuclei and elastic fibers it is covered by thinned epidermis without rete ridges or appendages (Fig 155) If the fibrosi is insignificant the scar rises but little above skin level But if it is very pronounced and exceeds the traumatized area then characteristic bizarre stripes and oblong extensions develop suggestive of the claws of a crayfish This appearance explains the name *keloidal scar* (Greek *chele* crayfish claw) given to these lesions If such fibromas with a thin covering of epidermis develop without preceding injury one uses the term *genuine or spontaneous keloid* In most cases it cannot be determined with certainty whether or not insignificant trauma *e g* a suppurative folliculitis, might have preceded the keloid For this reason the distinction between keloidal scars and true or idiopathic keloids is vague

In extensive scars, both atrophy and hypertrophy may coexist side by side (Fig 156) Often a scar is first hypertrophic then flattens in the course of months and years, assuming an atrophic character a development which is cosmetically desirable and can be enhanced by cautious radium X ray or grenz radiations.

The smoothness and gloss of the surface of the scars is subject to various changes Sometimes remnants of the rhomboidal skin relief may be retained and scales resembling ichthyosis may form (Fig 157) In other cases keratin formation may become so pronounced that yellow horny masses pile up Sometimes flaccid blisters filled with serous or hemorrhagic fluid form on fresh scars Such scars are popularly called *active* or *working* scars Some other scars because of their reduced elasticity tear forming painful fissures In other scars, especially those under tension over bony prominences the blood supply in some areas is sufficiently disturbed to cause necrosis, followed by slow healing scar ulcers Shrinking or contraction of scar tissue after considerable time may also cause disturbances of motility and various disfigurements (ectropion pterygium web formation microstomia)

It is difficult to define the differentiation between atrophy and atrophic scar The *genuine* (primary) *atrophies* are true atrophies in the strictest sense They are caused by a regressive process without preceding inflammation or other pathologic change The best known example is senile atrophy of the skin (Fig 158) In old age the entire skin may become atrophic i e smooth glossy largely hairless excessively easy to shift and because of this latter quality

seen on the fingertips in Raynaud s disease (Fig 152) In more severe cases all fingers may disappear or the stubs may fuse to leave an ugly stump (bullosis spontanea congenita Siemens Fig 153) It is characteristic of the lupus form of tuberculosis that it destroys only the skin and cartilage of the nose leaving a tipless nose ("worn-off nose) Nasal involvement in syphilis is mostly localized in the bony parts causing the bridge of the nose to collapse but leaving the tip relatively intact (saddle nose)

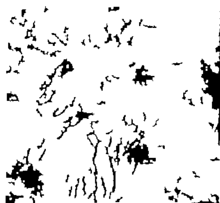


Fig 150 — Atrophic scar with telangiectasias (X ray burn)



Fig 151 —Poikilodermic atrophy (radium burn)

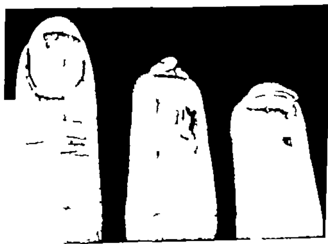


Fig. 152.—Mutilation of the fingertips (R yn d disease)

In a certain way scars can be considered as types of lesions in a category between those lesions which are always raised *above* and those which are always sunk *below* the level of the skin. If the cutaneous connective tissue is thinned, a depression, no matter how minimal will result. This is the *atrophic scar* (Fig 140 p 88) Frequently at other times in scar formation a hypertrophic reaction of connective tissue ensues causing the scar to bulge like a tumor This results in a *hypertrophic scar* (Fig. 154) The hypertrophic scar is a fibroma (more properly a *collagoma* because only the collagenous fibers are thickened) which is poor in blood vessels nuclei and elastic fibers It is covered by thinned epidermis without rete ridges or appendages (Fig 155) If the fibrosis is insignificant the scar rises but little above skin level But if it is very pronounced and exceeds the traumatized area then characteristic bizarre stripes and oblong extensions develop suggestive of the claws of a crayfish This appearance explains the name *keloidal scar* (Greek, *chele* crayfish claw") given to these lesions If such fibromas with a thin covering of epidermis develop without preceding injury one uses the term *genuine or spontaneous keloid* In most cases it cannot be determined with certainty whether or not insignificant trauma e g a suppurative folliculitis, might have preceded the keloid. For this reason the distinction between keloidal scars and true or idiopathic keloids is vague

In extensive scars, both atrophy and hypertrophy may coexist side by side (Fig 156) Often a scar is first hypertrophic then flattens in the course of months and years, assuming an atrophic character a development which is cosmetically desirable and can be enhanced by cautious radium X ray or grenz radiations

The smoothness and gloss of the surface of the scars is subject to various changes Sometimes remnants of the rhomboidal skin relief may be retained and scales resembling ichthyosis may form (Fig 157) In other cases, keratin formation may become so pronounced that yellow horny masses pile up Sometimes flaccid blisters filled with serous or hemorrhagic fluid form on fresh scars. Such scars are popularly called *active* or *working* scars. Some other scars because of their reduced elasticity tear forming painful fissures. In other scars especially those under tension over bony prominences, the blood supply in some areas is sufficiently disturbed to cause necrosis, followed by slow-healing scar ulcers Shrinking or contraction of scar tissue *after considerable time* may also cause disturbances of motility and various disfigurements (ectropion pterygium or web formation microstomia)

It is difficult to define the differentiation between atrophy and atrophic scar The *genuine* (primary) *atrophies* are true atrophies in the strictest sense They are caused by a regressive process without preceding inflammation or other pathologic change The best known example is senile atrophy of the skin (Fig 158) In old age the entire skin may become atrophic i.e smooth, glossy largely hairless, excessively easy to shift and because of this latter quality

seen on the fingertips in Raynaud s disease (Fig 152) In more severe cases all fingers may disappear or the stubs may fuse to leave an ugly stump (bullosis spontanea congenita Siemens Fig 153) It is characteristic of the lupus form of tuberculosis that it destroys only the skin and cartilage of the nose leaving a tipless nose ("worn-off nose) Nasal involvement in syphilis is mostly localized in the bony parts causing the bridge of the nose to collapse but leaving the tip relatively intact (saddle nose)



Fig 150 — Atrophic sca ith telangiectasias (X ray burn)



Fig 151 —Poikilodermic trophy (radium burn)

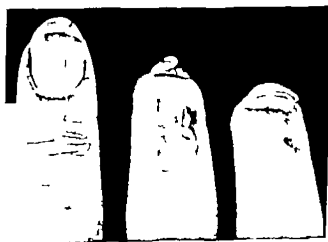


Fig. 152—M tilation of the fingertips (Ra naud s disease)

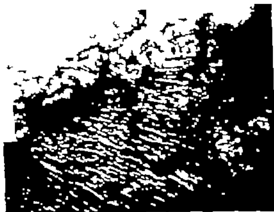


Fig. 156.—Atrophic and hypertrophic scars side by side (after treatment of lupus vulgaris with the diathermy loop)



Fig. 157.—Atrophic scar with ichthyosiform scales and hyperpigmented skin



Fig. 158.—Primary atrophy (atrophia senilis)



FIG. 153.—Mutilation of the entire hand (bullosis spontanea congenita Siemens)



FIG. 154.—Hypertrophic scar (after surgery and suture)

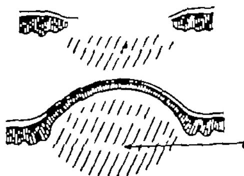


FIG. 155.—Atrophic and hypertrophic scars *a* blood vessels and elastic fibers decreased *b* same but with increased elastic fibers.

true that this anomaly often affects the epidermis also which becomes extended and loses its rete ridges. Thus the anelastic foci acquire a great similarity to ordinary atrophies, their surfaces being smooth and glossy and easily thrown into wrinkles which, however bulge a little (Fig. 161) The best known examples of anetodermatic atrophy are the striae of pregnancy which may occur as long bands as well as diamond-shaped figures (Fig. 162) Sometimes the affected areas of the skin look somewhat depressed sometimes if the skin has lost its normal tension they bulge out and form something like a connective

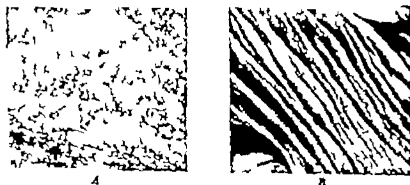


Fig. 159. A, smooth atrophic senile skin. B, wrinkling on pinching the skin together

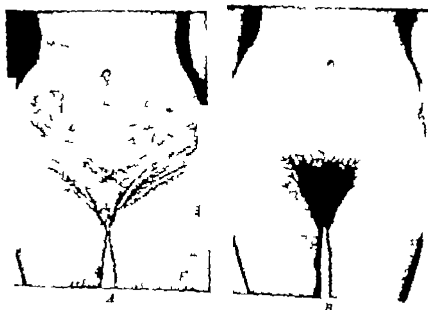


Fig. 160. A, defluvium in sclerosis. B, regrowth of hair after healing

wrinkled like crushed cigarette paper As always when large skin areas become thinned, the blue deeper veins and the yellow tendons may become more or less faintly visible There is no anatomical difference between the primary atrophies and the scar atrophies a fact which is expressed clinically by the same degree of smoothness glossiness and wrinkling (Fig 159 *A*, *B*) The *atrophies by defect* (secondary) may be subdivided into the posttraumatic or postulcerative types (the actual scar atrophies) and all the other secondary atrophies which appear as the final stages of interstitial pathologic processes without preceding defects of the skin (lupus erythematosus scleroderma lichen planus atrophicans alopecia atrophicans)

The same interstitial processes which cause secondary atrophies may occasionally lead to a fibrous toughening and hardening *sclerosis* (fibrosis) of the skin In this case the skin as in sclerotic scars is difficult to fold and is immobilized boardlike and firmly fixed to its base Atrophy and sclerosis may occur next to each other and in the same area successively More important than the *genetic* classification of atrophy into primary and secondary types is the simple delineation and terminology of the different *morphologic* types. In this respect it seems best to differentiate atrophia *simplex* (common atrophy and scar atrophy) atrophia *variegata* (poikiloderma) atrophia *fibrosa* (sclerotic atrophy) atrophia *fibromatosa* (hypertrophic scar and keloid) and atrophia *anetodermatica* (anelastica see below)

It is characteristic of a number of diseases such as tertiary syphilis lupus vulgaris some pyodermas (ecthyma furunculosis) variola and acne that they regularly or at least frequently lead to scarring from ulceration suppuration and necrosis On the other hand a few skin diseases are characterized just by the fact that scar formation develops *without* preceding visible destruction of tissue This is particularly true of lupus erythematosus scleroderma favus of the scalp and alopecia atrophicans Thus lupus erythematosus is indeed a scaling *erythema luposum* i e an erythema which leads to scar formation (*lupus* Latin for wolf meaning an eating destructive process) On the other hand there are dermatoses which practically never form scars. This is true and of diagnostic importance especially in eczema and psoriasis even though shedding of hair may occur in these diseases In itching chronic eczemas hairs may be rubbed off especially from the lateral parts of the eyebrows (Fig 97 p 68) but the follicles remain intact so that hair can grow again after healing of the inflammatory process (Fig 160 *A* and *B*)

If local degenerating changes appear in the cutis involving the elastica in a major way areas will develop into which the palpating finger so to speak falls as if into a pit If such gaps are small and round they give a feeling similar to that of pressing an electric push button There is a round center which can be easily pushed below the level of the surrounding skin This connective tissue atrophy involving the elastica (*anelasty*) is termed *anetoderma* (soft skin) It is



Fig. 161. Anetodermic atrophy roundish (anetoderma maculosum)



Fig. 162.—Anetodermic atrophy linear (striae gravidarum)



Fig. 163.—"Herniated" anetodermic atrophy (acne vulgaris)



Fig. 164.—Atrophy of the subcutis (lipodystrophy)

tissue hernia (Fig 163) At the start, their color is erythematous, livid red or bluish later this is changed usually to an iridescent white. If the epidermis is normal and the atrophic process takes place in the subcutaneous fat, deep indentations covered with a taut epidermis (Fig 164) may form.

Now we shall discuss those lesions which we call *deposits* or *scoriae* (Latin for slags) because they can be removed without injury

Since new epithelial cells are continuously being formed in the deepest layers of the epidermis the end product of their development i.e the dead dry horny scales, must be shed from the surface at an equivalent rate. However this normal shedding of keratinized cells can be observed only under artificial conditions e g the accumulations of horny material under a plaster cast kept in place for a long time This ordinary invisibility of physiologic desquamation is easily explained because the sheddings are very minute particles representing so to speak a *desquamatio insensibilis* A desquamation which is plainly visible is always pathologic It occurs most frequently as the consequence of inflammation

Three different anatomical processes may lie behind *increase and thickening of the horny layer* either with or without noticeable exfoliation (1) hyperkeratosis (2) parakeratosis and (3) dyskeratosis.

In the case of *hyperkeratosis* the horny layer is augmented but otherwise looks normal The underlying keratohyaline layer is also thickened or at least normal Hyperkeratosis may come about by *increased proliferation* of cells. In this case the rete and keratohyaline cells are also increased in number conditions termed *acanthosis* and *granulosis* This type of hyperkeratosis is called *proliferation hyperkeratosis* and the callus is a good example of it In other cases the thickening of the horny layer is caused by a *decreased shedding* Here insensible desquamation apparently fails to take place properly because the horny cells stick abnormally tightly together as well as to their base. This is called *retention hyperkeratosis* In this case the shedding of surface horny material is retarded and eventually occurs in larger accumulations which thus become visible as scales (e g ichthyosis) There is no essential change in the rete.

Parakeratosis which is the most common anatomic feature in the horny layer associated with scale formation is always caused by an inflammation of the papillary layer of the cutis Inflammatory processes in the cutis always disturb keratinization so that in the uppermost layers of the epidermis the formation of keratohyaline granules is absent in stretches in which therefore no stratum granulosum exists Thus in such areas keratin is formed directly from the epidermal cells of the rete malpighii with skipping of the keratohyaline phase. In these abnormally formed horny cells the nuclei remain preserved and can be stained Possibly the inflammatory hyperemia causes better nutrition of the skin with subsequent increased proliferation of rete and horn cells Obviously the coherence of the horn cells is also altered in the parakeratotic process. On

lentil-sized lesions of psoriasis often appear as smooth brownish red hard prominences which may be mistaken for papules until gentle scratching suddenly makes them light up with a silvery-white color This phenomenon results because scratching permits air to enter between the horny cells and cause the brownish, smooth material to fall apart in mica like scales. This has been called the *candle-drop sign* (Fig 166 *A* and *B*) In parapsoriasis guttata the horny layer which covers the lentil-sized lesion does not disintegrate into silvery platelets on scratching but rather comes off as a whole lamella (wafer-like desquamation, Fig 167) The latent desquamation is again different in tinea versicolor In this disease, which forms brown maplike spots on the chest and back the fungi grow between the horny layer and the rete so that the horny layer is more or



Fig 165 —Central desquamation (pityriasis rosea near na el)

less detached from its base but is still coherent within itself If one scratches the apparent macule in this disease with a vigorous stroke one tears off lentil sized ragged pieces which, however are often still adherent at one end (*wood shaving-like desquamation* Fig 168 *A* and *B*) It is true that one may elicit the same phenomenon by scratching the normal skin but it is not so easily accomplished, and the scales are not so typically big as in tinea versicolor Scales are most frequently encountered in the erythemato-squamous dermatoses, inflammatory diseases whose main symptoms are redness and scaling The types of scaling in these conditions may be pityriasiform psoriasiform and sometimes cuticular Transitions between these types also occur If an erythemato-squamous eruption occupies very large areas of the body surface i.e. if it is diffuse and generalized or universal it is spoken of as *erythrodermatic scaling*

the surface these parakeratotic cells form a brittle splintery covering which comes off in small or larger fragments sometimes resembling dust or mica platelets and sometimes forming small membranes (squamous eczemas, psoriasis, superficial trichophytosis etc.) The presence of parakeratosis can often be deduced from the accompanying inflammation but certain differentiation of hyperkeratotic and parakeratotic desquamations is possible only by histologic examination This is particularly true because hyperkeratosis and parakeratosis frequently occur together in the same dermatosis (clavus keratosis follicularis pilaris parapsoriasis)

Dyskeratosis is a form of keratinization in which there appear individual large round inflated looking cells with keratohyaline granules and frequently with a double horny membrane These cells later shrink to irregularly shaped but still nucleated horny granules (grains which can also be called *chondrocytes* from Greek *chondros* grain) This faulty keratinization is encountered most frequently in Darier s disease and in Paget s and Bowen s epitheliomas. It also occurs though less often in molluscum contagiosum other epitheliomas, and verrucae Clinically dyskeratosis causes either brownish crustlike grains of horny substance or just ordinary desquamation The diagnosis of dyskeratosis can be made with certainty only microscopically

Increased and pathologically altered keratinization sometimes results in the formation of coherent firm horny masses but more frequently in desquamation *Squamae* scales are independently exfoliated platelets consisting of groups of coherent horny cells. They occur in any size from fine flourlike dust to thick laminated disks and extensive parchment like sheets. The following terms are used to describe scales *pityriasiform* (branny) *psoriasiform* (brittle platelets of several loose layers) *ichthyosiform* (like fish scales) *cuticular* and *lamellar* (thin relatively large flakes) *membranous* or exfoliative (large sheets) *keratotic* (composed of horny masses) the latter may again be subdivided into *callous* or tylotic *granular* *hystrix like* (from Greek *hystrix* 'porcupine) and finally scale formations resembling little horns (squamae cornutae)

A special group is formed by *follicular* scale formations which may be subdivided into *keratotic plugs spines or filaments* and *lichenoid scales*

It should be emphasized that a characteristic scale formation can sometimes be recognized only after *scratching the lesion* In these cases the pathologically thickened horny masses are still coherent even though an increased fragility of the horny layer exists and becomes apparent on scratching This fragility however is not pronounced enough to manifest itself from the ordinary insults of movement and rubbing by garments Such *latent desquamation* (status predesquamatosus) is regularly found in the early stages of the lesions of pityriasis rosea We may then see only a lentil sized red spot with a slightly brownish center of the size of a large pinhead which will desquamate in branny particles after scratching (*central scale formation* Fig 165) Also fresh not more than

Such eruptions are encountered in very extensive skin diseases (generalized psoriasis, generalized squamous eczemas) and are also characteristic of the so-called erythrodermas. There are *secondary erythrodermas* which are rare complications of dermatoses usually occurring only in circumscribed forms and also *primary erythrodermas* which form a separate class of diseases. Erythroderma is always universal or almost universal. "Erythroderma" means red skin, but, as already pointed out, the involved skin is not only red but also scaly Thus the disorder might better be called an *erythrosquamia*.

We speak of branlike, *pityriasiform desquamation* (*pityron* Greek for "bran") if the scales are very small, comparable to dust, flour or bran (Fig 169) This type of scaling is found mainly in squamous eczemas, in pityriasis capitis (dandruff) in parapsoriasis in superficial trichophytosis and in erythrasma.

If the scales are somewhat bigger mica-like and iridescent because of their ample air content, we speak of *platelet-like* or *psoriasiform desquamation* (Fig 170) As the name indicates, this type of scaling is found mainly in psoriasis, but it may also occur in a great variety of other skin diseases. Therefore one speaks of psoriasiform eczemas psoriasiform syphilis, psoriasiform lupus, etc. Psoriasiform desquamation makes its appearance wherever layers of horny matter have piled up and have become dry and brittle so that air can easily enter between the layers Sometimes the desquamation is brittle on the surface only but is firmly attached below a combination which causes white silvery hard spots that cannot be easily scratched off (Fig 171) If the accumulation of keratinous substance is very heavy and the individual layers are bound together by a small quantity of exudate the scales will not crumble off Thus very thick masses may pile up. This is frequently the case in lesions which periodically enlarge in area, so that the initial mass of scales is lifted up by a new larger one and this again by another one of still larger circumference By this method of growth, obtuse cone-shaped piles of scales with horizontal layers reminiscent of oyster shells develop (psoriasis ostracea, ulcero-squamous syphilis, ulcerating leprosy) This *oyster shell-like desquamation* (Fig 172) is such a striking phenomenon that dermatologists of former generations considered it an entity and called it *rupia*

Psoriasiform scales on the hairy scalp will *not* easily break down to a fine powder as in pityriasiform dandruff (pityriasis capitis) but rather cling to the hair like large platelets (Fig. 173) They may sheathe the hairs (Fig 175) or they may look much like asbestos (*asbestos-like desquamation* Fig 174) (Fig. 175)

It is characteristic of some desquamations to form regular smooth roundish or more polygonal, lentil-sized and also larger shields which are attached in the center and loose at the edges, where they become most visible (Fig 176) These shieldlike or fish-scale like *ichthyosiform desquamations* are found especially in ichthyosis, in which, in the affected areas, the shields are arranged in



A



B

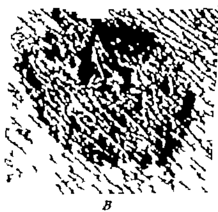
Fig. 166.—Latent desquamation (*A*) before and (*B*) after scratching: candle-drop phenomenon (erupting psoriasis)



Fig. 167.—Wafer like desquamation (parapsoriasis guttata of the elbow)



A



B

Fig. 168.—Wood-shaving-like desquamation (*tinea versicolor*) (*A*) before and (*B*) after scratching

Such eruptions are encountered in very extensive skin diseases (generalized psoriasis, generalized squamous eczemas) and are also characteristic of the so-called erythrodermas. There are *secondary erythrodermas* which are rare complications of dermatoses usually occurring only in circumscribed forms and also *primary erythrodermas* which form a separate class of diseases. Erythroderma is always universal or almost universal. "Erythroderma" means red skin but, as already pointed out, the involved skin is not only red but also scaly. Thus the disorder might better be called an *erythrosquamia*.

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Fig. 169 —Branlike (pityriasiform) desquamation (parapsoriasis areata)

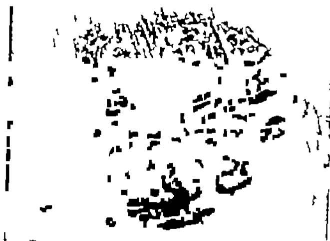


Fig 170 —Psoriasiform desquamation, loose (psoriasis)



Fig 171 —Psoriasiform desquamation, ad herent (psoriasis)



Fi 1 2 —Oyst -sh ll ffi desquam tion rupia (psoriasis



FIG. 173.—Loose psoriasiform scales in the hair (psoriasis)



FIG. 174.—Asbestos-like desquamation (psoriasis capitis)



FIG. 175.—Hairs ensheathed by scales (psoriasis)

parallel rows (Fig 177) or in diamond patterns. They may also be encountered in eczemas and erythrodermas A desquamation which is somewhat similar is *état craquelé* Here only the thin horny layer cracks forming a coarse network of superficial hair thin rhagades which is faintly reminiscent of the glaze of old Chinese pottery (*cracking desquamation* Fig 178) In contrast to these scales, which are attached *in the center* some adhere on one side while the other side is loose ragged or even a little rolled up These are scales which before being shed still cling for a while to the body like a hangnail or cuticle I call this common type of scale formation *cuticular desquamation* (Fig 179) Sometimes these cuticles may be very large (giant cuticulae Fig 180) This cuticular desquamation is often associated with the pityriasiform and psoriasiform types, but it may also occur alone e g in eczemas and mycoses between the toes. A peculiar form of cuticular desquamation is the wood shaving like desquamation in tinea versicolor which has been described before.

Other special forms of scale formation are the *cracklike desquamations* caused by the cracking-open of scabies burrows (Fig 181) which are diagnostic of scabies and the collar like desquamations in which after exfoliation of the centers of its lesions *ring shaped collars remain* which are identical with the previously described collarettes Such scale collars (*collarettes* coronellae or corollae) are mostly found in eczemas, in superficial mycoses (Fig 182) in tinea imbricata in secondary syphilis, and in the characteristic lesions of pityriasis rosea (Fig 183) They may also appear as giant collarettes (Figs. 184 and 185) or as several concentric rings (Fig 186) If loose exfoliating scales reach fingernail size or more one can speak of *leaflike* or *lamellar desquamation* (Fig 187) This variety of desquamation occurs in pemphigus foliaceus and in some erythrodermas If a new scale forms at the base of each scale before the old one becomes completely detached accumulations suggesting piecrust develop frequently accompanied and enhanced by exudative processes

If the horny layer comes off in still larger sheets I speak of *membranous desquamation* or *peeling* (Fig 188) Peeling may be artificially obtained by peeling agents (salicylic acid resorcinol) and entire casts of fingers palms and soles may be shed but it may also be observed in confluent bullous eczemas, in erythrodermas and in dermatitis exfoliativa (membranosa) neonatorum Scales may at times acquire a special aspect by virtue of a high *sebum content*. This is a fairly regular occurrence in the pityriasiforum desquamations of the scalp the center of the face and the central areas of the chest and back (anterior and posterior sweat furrows) the sebaceous glands of these areas being especially big and numerous Here one refers to *seborrheic desquamation* (seborrhea flow of sebum) Seborrheic scales are yellowish to dirty yellow dull and feel greasy If crushed between cigarette papers they leave a grease spot They may contain up to 30 per cent lipid substances Squamous eczemas of regions with large sebaceous glands, such as the scalp and the central part of the face and chest produce particularly fatty scales. Therefore they are called *seborrheic eczemas*



Fig. 176.—Ichthyosiform desquamation (ichthyosis vulgaris of the lower leg)



Fig. 177.—Ichthyosiform desquamation (ichthyosis congenita partially healed, abdomen)



Fig. 178.—*Craquelé* pattern desquamation (irritation from sulfur)

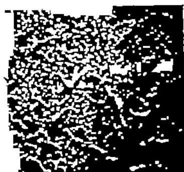


Fig. 179.—Cuticular desquamation (ichthyosis vulgaris)



Fig. 180.—Cuticular desquamation (eczema squamosum)



Fig. 181.—Cracklike desquamation (scabies burrow on heel)



Fig. 182.—Collarlike desquamation (small collarettes, moniliasis on coccyx)

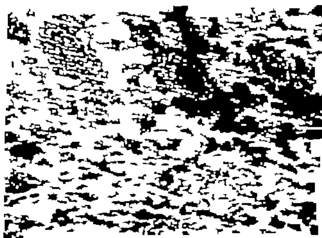


Fig. 183.—Oval collarette (pityriasis rosea)



Fig. 184.—Large collarette (eczema vesiculo-squamosum)

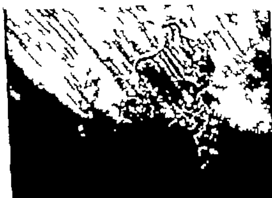


Fig. 185.—Giant collarette (tinea superficialis pedis)



Fig. 186.—Concentric collarettes (erythema annulare squamosum Siemens-Jagtman)



Fig. 181.—Cracklike desquamation (scabies burrow on heel)



Fig. 182.—Collar like desquamation (small collarettes moniliasis on coccyx)

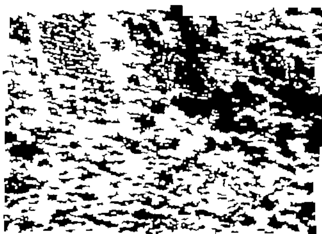


Fig. 183.—Oval collarette (pityriasis rosea)

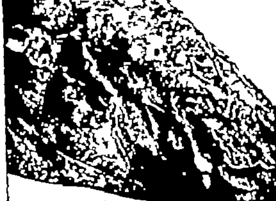


FIG 187 —Lamellar desquamation (eczema erythrodermaticum on the instep area)



FIG 188 —Membranous desquamation (peeling) in eczema universale



FIG 189 —Callous scale formation, papular (keratosis palmo-plantaris papulosa)

An inflammation which causes parakeratosis frequently entails exudative processes also so that the accumulated deposits consist of scales mixed with *serum* or *pus* These exudate-containing scales *squamae exudatrae* also look dull, yellow or brownish yellow and feel moist and sticky Formerly they were classified as a lesion bearing the name crusta lamellosa *crusted scale* They are most frequently found in pemphigus foliaceus but also occur in many desquamating eczemas Besides sebum and sero-purulent exudate scales may be saturated with *sweat saliva* or other liquids so as to cause a state of *maceration*. This occurs especially where skin touches skin (axillae groins intergluteal fold) and on the mucosae On the latter the macerated horny thickenings which appear white are called *leukoplakias* Keratoses of mucosal surfaces should not be confused with mucosal plaques which consist of macerated epithelium or with pseudo-membranes (see below) One may refer to *keratotic desquamation* (squamae corneae) if hard firmly adherent keratinous masses are accumulated on the skin These tylotic or *callous scale formations* may be in the form of pinhead-sized nodules which after a time often lose their keratotic centers (keratosis palmo-plantaris papulosa Fig 189) They may also result in lentil to silver-dollar sized horny pads (calluses) on palms and soles or may tend to cover the skin in a more diffuse manner (callous eczemas, keratosis palmo-plantaris diffusa) giving the affected areas a yellow waxlike color and blurring the normal skin surface relief (Fig 190) *Callous horn formations* have the color of horny materials namely yellow to yellowish brown often greenish black if of long duration and milky white if macerated (sweaty hands) Their surfaces may be smooth without visible exfoliation (Fig 190) pitted (Fig 191) covered with more or less loose scales, or verrucous (Fig 192) They may also show cracks (Fig 193) or pits surrounded by collarettes. If there are no cracks, the surface relief may be coarsened and may exhibit deep furrows, so that one is reminded of lichenification (Fig 194) However in lichenification the thickening of the skin which is the cause of the coarse relief is situated in the rete (acanthosis) and in the cutis (infiltrate) while in keratosis it is in the horny layer The two phenomena should not be confused with each other or with the insignificant accentuation of skin relief found in macerated sweaty hands (Fig 195)

The lack of elasticity in callous skin areas may cause superficially squamous (Fig 196) or sometimes deeper cracks (*fissures* Fig 197) which penetrate into the papillary body eventually causing severe pain and offering a portal of entry for many kinds of infections.

The *location* of calluses is frequently very typical because they are likely to form in places which are exposed to constantly repeated pressure and rubbing Knowledge of their exact location and shape frequently permits conclusions to be drawn as to the activity or vocation of the patient (rowing cleaning woman cobbler milker sculptor) or in some countries the type of clothing worn (wooden shoes Fig 191)



Fig. 194.—Callous scale formation, diffuse, without cracks but with coarsened relief (keratosis palmo-plantaris diffusa)



Fig. 195.—Deepened relief by maceration from sweat (hyperhidrosis palmo-plantaris)



Fig. 196.—Squamous fissures in callous scale formation (eczema callosum)



Fig. 197.—Excoriative fissures in callous scale formation (pityriasis rubra pilaris)



Fig. 190.—Callous scale formation diffuse, with blurred relief (keratosis palmo-plantaris diffusa)



Fig. 191.—Callous scale formation, nummular with pitted surface (callus from wearing a wooden shoe)



Fig. 192.—Verrucous desquamation (eczema keratosum)



Fig. 193.—Callous scale formation diffuse with cracks (keratosis palmo-plantaris diffusa)



Fig 194.—Callous scale formation, diffuse, without cracks but with coarsened relief (keratosis palmo plantaris diffusa)

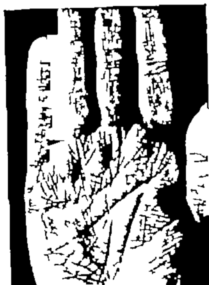


Fig 195.—Deepened relief by maceration from sweat (hyperhidrosis palmo-plantaris)



Fig 196.—Squamous fissures in callous scale formation (eczema callosum)



Fig 197.—Excavative fissures in callous scale formation (pityriasis rubra pilaris)

Besides these callous horn formations which are only little raised above the skin we may also find firmly adhering mostly yellow brown keratoses which are pointed and protrude conically such as the cornu cutaneum (horn-shaped scales squamae cornutae Fig 198) Molluscum contagiosum on occasion may also bear a tiny spine-shaped horn With the callous scale formations and the cornua cutanea may be grouped the keratotic vegetations occurring either in circumscribed areas or spread widely over the body surface If the individual protrusions tend to be round one may speak of *squamae granulosae* (Darier's disease lichen planus ichthyosis) If these granules are larger their sides become compressed and they present mosaic or cobblestone like arrangements (Fig 199) If the prominences are long and pointed (ichthyosis hystrix verrucae vulgares verrucous nevi keratotic eczema) one may use the term *squamae hystriciformes* which makes use of an old fashioned exaggerated comparison

Finally the *follicular desquamations* may be mentioned as a special clinical group They also frequently have a keratotic character i e form hard granules which cannot be removed from the skin without force This is certainly true of those diseases in which comedo-like pinhead sized or larger brown to black horny plugs fill the follicles (keratosis follicularis acneiformis pityriasis rubra pilaris Fig 200) or protrude as dark granules or little balls from the follicles (lupus erythematosus Fig 201) The latter may drop out leaving pitted follicular scars (Fig 202) If the accumulations of horny substance are smaller they are just as difficult to remove In the earlier stages they are of lighter color and more spiny so that an area of the skin in which all follicles are involved feels like a grater (lichen ruber acuminatus tar acne Fig 203) If the follicular scales are still thinner they may be found in each follicle as short whitish filaments which stick out from the surface more or less perpendicularly (keratosis follicularis spinulosa Fig 204) If they are not pointed but rather roundish they may look like hard nodules (keratosis follicularis lichen pilaris) All these plugs spines and filaments may take the place of hairs or such horny elements may be present next to hairs (alopecia atrophicans lupus erythematosus) Sometimes the skin *between* the follicles is covered with a whitish chalklike to blackish horny layer If one carefully lifts this coherent scale cover small pointed horny pegs which correspond to the follicle openings may adhere to the undersurface of the scale This type of scale is characteristic of lupus erythematosus

Crusts (crustae) are deposits which consist of dried secretions or in the case of necrotic crusts of dead tissue Accordingly we differentiate *serous purulent bloody* and *necrotic crusts* Besides the ordinary components of the respective secretions from which they originate (leukocytes erythrocytes etc) they often contain other additions like scales, bacteria and dirt In some cases the crust is covered by a horny lamella (*crusta subcornealis*) which is later shed together with it. This occurs in those cases where the exudates of vesicles or pustules dry before their tops burst

Naturally the appearance of crusts depends on the nature of the dried secretion. *Serous* crusts are translucent, like the resin gum of cherry trees, and yellowish brown honey-colored or varnish-like if the crusts are thin. *Pus crusts* are turbid, opaque whitish yellow yellowish green or even brown if there is an admixture of serum and blood (Fig 205) *Bloody* crusts are red purplish or almost black and also opaque *Necrotic* crusts are dirty yellow as long as they are moist and deep black when they are dry In the latter case they are called *eschars* Frequently crusts are heterogeneous, especially sero-purulent and ad



Fig 198 —Hornlike desquamation (cornu cutaneum)

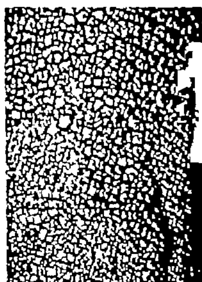


Fig 199 —Granular cobblestone like desquamation (ichthyosis nigra granulosa)



Fig 200 — Follicular keratosis, plugs (keratosis follicularis on the knee)

mixed with more or less blood or they may be serous at the start and become purulent later because of secondary infection

Crusts may be very small or even punctate This latter type is frequently encountered in scratch marks when very small papules or vesicles are scratched open (Fig 206) In such cases the tiny dark red points are frequently so small that at first glance they may easily be mistaken for petechiae However palpation soon clarifies the situation because dried up secretions feel brittle If secondary pyogenic infection supervenes (impetiginization pyonization) scratch marks may develop big sero-purulent crusts which coalesce In the beginning



Fig. 201 —Follicular keratoses, granules (lupus erythematosus)



Fig. 202 Follicular keratoses, granules and follicular pits after the dropping-out of the granules (lupus erythematosus)

there is always some remaining liquid exudate beneath a crust If this exudate is copious one can see pus well up under the edge of the crust if pressure is exerted upon it Owing to moisture warm temperature and occlusion such exudates offer very favorable conditions for the growth of saprophytes and pyogenic cocci If such infected crusts are not removed ulcers are liable to develop beneath them which in turn may lead to lymphangitis and in infants, even to sepsis. In any case suppuration at their bases makes crusts more succulent and thicker Therefore thick crusts are always indicative of secondary pyogenic infection (impetigo and ecthyma)

Crusts may cover skin changes which lie more or less in the level of the skin (erosions and excoriations) they may top exudative prominences (granulations, syphilitic papules carcinomas) or they may fill defects In the first and

second cases they will rise above the surface of the skin as in impetigo while in the latter case they may be imbedded in the skin often level with its surface as is frequently the case in ecthyma (Fig. 207) The surfaces of crusts are smooth or crumbly Crusts usually feel hard and sometimes even like bits of broken glass, which makes the diagnosis of small crusts easier They may be attached firmly or loosely They are easy to remove if exudation from underneath still continues. When a new horny layer forms underneath a crust it drops off spontaneously In order to arrive at a diagnosis, it is sometimes necessary to make the base of a crust visible by removing the crust. This is most gently done by

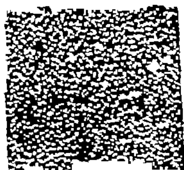


FIG 203.—Crater-like follicular keratoses after treatment with tar



FIG 204.—Follicular keratoses with little spines (Keratosis follicularis spinulosa)

dressing with a copious covering of thick ointment (*e.g* ung diachylon) and, if necessary with the addition of salicylic acid In hairy areas, oil or cod-liver oil is brushed onto the lesion which is then covered with oil-soaked gauze. Necrotic scabs which can hardly be removed prior to their demarcation, may sometimes be loosened with 5 per cent hydrogen peroxide

Some dermatologists differentiate *necrotic crusts* (crustae necroticae) as eschars because they do not consist of dried fluid but of dead tissue They are yellow-brown to deep black, are situated in or below the level of the skin, are firmly attached to their base and are shed only after considerable time by way of a demarcating inflammation (scabs of burns, herpes zoster) If they are small and round, they resemble dried pustules (crusta pustuloides Fig 62 p 54) or may easily be confused with pustules (acne necroticans, papulonecrotic tuberculids) True crusts can form only where the seeped-out secretion really can

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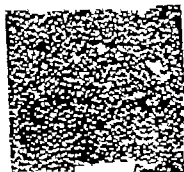


Fig. 203 —Crater like follicular keratoses after treatment with tar



Fig 204 —Follicular keratoses with little spines (keratosis follicularis spinulosa)

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Fig 205 —Crust serous and purulent (Impetigo streptogenes)

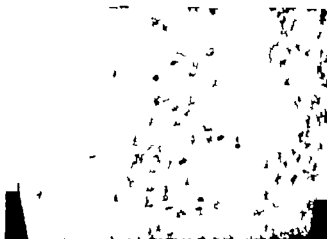


Fig 206 —Crusty scratch marks, very small (eczema papulosum)

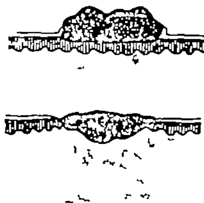


Fig 207 —Superficial (impetigo) and deep (ecthyma) crusts

dry and not where desiccation is hampered by intertriginous contact of skin areas or by moist dressings or on mucosae Then instead of a crust a moist, sticky disintegrating layer of tissue forms which may be purulent fibrinous (diphtheroid) ragged, or necrotic. It can be easily scraped off at the surface but is often firmly adherent below and cannot be removed without bleeding Such changed tissues are called *pseudo-membranes*

Besides scales, crusts, and pseudo-membranes we occasionally find on the skin *deposits which are alien to the body* This is especially the case in fungus diseases. The mealy sheaths surrounding the hairs in microsporum infections (Fig 208) the yellow scutula of favus (Fig 209) and the mycelia of tinea versicolor growing under the horny layer (Fig 168 p 102) have already been mentioned. There are also the granular deposits on the hairs in piedra and trichomycosis palmellina which consist of fungous elements and cocci (see p. 166) and, finally the products of epizoa of the skin may adhere to the hairs, such as nits or their empty chitinous shells (Fig 283 p 168) of head and pubic lice. It hardly needs to be mentioned that dirt and remnants of dermatological medicaments may also cause a variety of deposits which frequently but by no means always, can be washed, scratched, or scraped off (corpora aliena)

In diagnosing skin eruptions, it should never be forgotten that in most of them not just one lesion but several different types of lesions may be present. The majority of eruptions are *polymorphic* and it becomes the task of the examiner to ascertain the nature of all types of lesions which are present. Therefore inspection of the entire skin surface of the patient is often necessary *Polymorphism* is most typical if an eruption is a *dynamic one (dynamikos* Greek for "vigorous or "in movement) meaning that its basic lesions evolve as is the case for instance in eczema, whose papules become vesicles by spongiosis. Scratching causes these vesicles to become excoriated and they form erosions with crusts, which, in turn after being shed, leave erythema and scaliness. Another example is acne, which starts as a comedo and by inflammation, becomes a papule. By abscess formation the papule turns into a pustule which, when it drains, becomes covered with a crust and finally leaves a follicular scar (Fig 210) In contrast to these dermatoses are the *static* (*statikos* Greek for "bringing to a standstill" also "standing still") eruptions, whose basic lesions always remain the same in type (e g. lichen obtusus, with its hemispheric almost permanent, papule) or change only within the natural phases of healing (e g pemphigus, the blister of which becomes an erosion, which transforms itself into a residual erythema with scaly collarette) Such eruptions are then either completely or essentially *monomorphic* It is true that their lesions when they become older may look different and less characteristic than at the start An example is lichen planus in which the *youngest and smallest* papules show most clearly the characteristic polygonal and plateau-like shape In all cases the diagnostician *must* single out the primary lesion and then follow



FIG 208.—Microsporon fungi on hairs



FIG 209 —Favus scut la f the glabrous skin.

FIG 210 —Polymorphism in acne omedo papule pustule scar

its transitions in other words he must observe the evolutionary changes in the lesion.

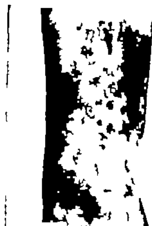
Polymorphism frequently comes about when an eruption appears in *crops* so that it presents lesions of different ages. This can be observed in varicella. Here the eruption period is 3–4 days, and young small and older larger lesions are present at the same time. This basis for polymorphism is even more true of strophulus, a condition in which only a few new lesions erupt daily. Here one may see at the same time erythematous wheals, wheals with central papules or vesicles, erosions following these vesicles, small crusts, and desquamating residual erythemas. To this same category also belong chronic diseases, such as chronic vesicular eczema, which only now and then develop an acute exacerbation. In this condition recent vesicles small crusts and erosions are found side by side with squamous or callous remnants of previous crops. This contrasts



Fig. 211 —Polymorphism from different stages of development among the individual groups of lesions (herpes zoster)

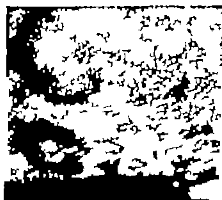
with diseases which run their course in *a single very acute eruption* such as variola, with all vesicles starting simultaneously and therefore exhibiting the same stage of development. A transitional position is occupied by eruptions with successive *crops in different sites* a good example being herpes zoster. Here the different groups of lesions because of their age differences, may exhibit very different lesions, such as urticarial erythema small vesicles large vesicles, crusts, and early stages of scars, while the elements of each group are in the same stage of development and are therefore identical (Fig. 211). In this case the individual groups of lesions are monomorphic but the whole eruption is polymorphic. Polymorphism may also occur if an eruption tends to develop differences in its lesions *according to the various body regions* where they appear. This is the case in lupus erythematosus, which usually produces erythematosquamous and atrophic lesions on the face but often only nondescript erythemas on the extremities (Fig. 212, *A B*). Another example is Darier's disease which





A B

FIG 212.—Polymorphism in different regions lupus erythematosus, (A) erythemato-squamous and atrophic in the face (B) purely erythematous on the arm





A B



C

FIG. 213.—Dermatitis herpetiformis Duhring with (A) urticarial erythemas on th upper arm (B) vesicles near the navel and (C) bullae all occurring simultaneously in the same patient.



FIG. 214.—Dermatitis herpetiformis, exclusively urticarial erythemas



FIG. 215.—Dermatitis herpetiformis, exclusively excoriated papules

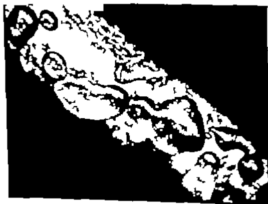


FIG. 216.—Dermatitis herpetiformis, exclusively bullae

causes brownish granular keratoses on the trunk but plateau like flat papules reminiscent of verrucae planae on the dorsa of the hands Conditions with different skin and mucosal lesions also belong to this group (lichen planus) Finally polymorphism may be encountered when one skin disease complicates another such as occurs in pyonization (impetiginized scabies) and eczematization (ulcers with eczematized surroundings)

By the way it should be kept in mind that the term *polymorphism* applies not only to a given dermatosis in *one* patient but also to those diseases which show one type of lesion in one case and another type in another case Thus *erythema multiforme* (multiform is synonymous with "polymorphous") may in one case—and this is not rare—be *monomorphic* consisting *exclusively* of erythemas or *exclusively* of vesicles yet the disease as such is multiform because it produces sometimes only erythemas, sometimes only vesicles, and sometimes erythemas raised erythemas and vesicles *simultaneously* By the same token Duhring s dermatitis herpetiformis may be termed *multiform* because we may observe in the same patient wheals papules, vesicles, and blisters simultaneously (Fig 213 1–C) It deserves this designation all the more because we may find in one patient only wheals besides occasional blisters (Fig 214) in another only (excoriated) papules (Fig 215) in a third, only vesicles and in a fourth only big blisters (Fig 216) Similarly one should understand the meaning of the name *tinea versicolor* (multicolored) While the fungus-containing spots which characterize this disease often have the same color at any given time in one patient they may variously appear white yellow or brown in different persons or even in the same person at different times.

In observing and evaluating lesions one should of course not forget to examine carefully *the skin between the lesions* which may be altered in many ways. It may be erythematous and swollen or the lesions may be set in pigmented or depigmented lichenified squamous or atrophic surroundings.

CHAPTER THREE

Extent, Shape, and Distribution

All the lesions of a skin disease together form what is called the *eruption* (rash) provided that it has the character of a process with a beginning, a climax, and an end. Mucosal eruptions are occasionally called *enanthems*, though this expression in the English speaking world is reserved mainly for the mucosal eruptions in systemic infections.

Eruptions on the skin and mucosae may run an *acute* course, meaning that they tend to spread and subside rapidly or, at any rate, change their appearance quickly. They may also be *chronic*, i.e., they may change only slowly either for better or for worse. The layman often thinks that "chronic" is synonymous with "incurable." Therefore, one should not use this expression in the presence of the patient, at least not without the necessary explanation of its meaning. Between the acute and chronic eruptions range the subacute, the moderately rapidly changing, and the chronic recurrent eruptions. The latter may even occur so that the individual attack may be acute and of short or relatively short duration while the disease itself, by its constantly recurring crops of lesions, may be long lasting (urticaria, secondary syphilis, chronic vesicular eczema). The term *acute exanthem* is used in a stricter sense for the acute infectious diseases of childhood which are associated with rashes (measles, scarlet fever, German measles).

The *extent* of eruptions may also vary widely. Many skin diseases are circumscribed, i.e., confined to one or several areas. Others occur regionally in certain usually symmetrical regions of the body. If eruptions start from small areas and gradually involve most parts of the body surface, we call them *generalized*. If either primarily or in the course of generalization they cover the entire or almost the entire skin, they are spoken of as *universal*. Even in eruptions of universal distribution some regions (palms, soles, axillae, groins, scalp) or a few irregularly distributed spots may usually remain free (psoriasis universalis). Within the involved areas the individual elements of skin diseases can show different types of arrangement. If we disregard the *solitary* lesions which are common in tumors and ulcers, the lesions may be grouped, disseminate, or diffuse. If the *grouped* lesions are vesicles, the term *herpetiform* is used, because the vesicles of herpes simplex characteristically occur in groups (Fig.

causes brownish granular keratoses on the trunk but plateau like flat papules reminiscent of verrucae planae on the dorsa of the hands. Conditions with different skin and mucosal lesions also belong to this group (lichen planus) Finally polymorphism may be encountered when one skin disease complicates another such as occurs in pyonization (impetiginized scabies) and eczematization (ulcers with eczematized surroundings)

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which are most frequently used are *miliary* (*milium* Latin for 'millet') *guttate* (*gutta* Latin for 'drop') *lenticular* (*lenticula* Latin for "small lentil") and *nummular* (*nummulus* Latin for "small coin") The word *discoid* is practically identical with *nummular* except that it is occasionally used for lesions which are larger than a coin. The fact that coins come in somewhat different sizes is no disadvantage but rather an advantage of this comparative terminology because lesions of larger than lentil size also mostly have different sizes. If lesions are a little larger than lenticular and ovoid they may be referred to as almond sized or almond-shaped. If there are only a few larger patches one calls the arrangement *en plaques*. If there are only single very large patches, the French use the term *en placards*. If a disseminated exanthem consists of *lenticular erythemas* it is called a *roseola* (typhoid fever syphilis). Disseminated lesions may be sharply separated in which case they are called *discrete*. On the other hand, they may also coalesce. Such *confluence* may come about by *peripheral growth* of the individual lesions so that they gradually touch one another merge, and then form extensive patches with polycyclic edges (Fig 219). Confluence may also develop by *apposition*. In this case the lesions become more and more numerous until finally no room is left between them. This occurs in diseases whose lesions have only a small capacity for growth and therefore remain small (lichen planus, lichenified eczemas, Fig 220). By apposition in this manner even universal dermatoses may develop (lichen planus). *Sometimes* eruptions may primarily involve large areas rather than discrete spots, or they may spread as a continuum (scarlet fever generalized eczema erysipelas). Eruptions which cover large areas in these ways are called *diffuse* (Fig 221).

The *shapes* of the lesions which compose eruptions may vary widely. Most frequently they are *round* or *oval*. This is particularly characteristic of acute erythemas and can be explained on anatomical grounds. Each individual erythematous spot indicates the area of vascularization supplied by *an arteriole* and thus represents a territory of direct blood supply. From the center of this area, however the erythema then spreads *evenly in all directions*. It by no means follows the lymphatics, which would create a spot with pseudopod-like extensions i.e. a dendritic shape (see below) nor does it follow the blood capillaries which also do not radiate in all directions from one point. It simply creeps along as if stimulated by something diffusing through the spaces between cells and connective tissue fibers in tissue fluid which is neither blood serum nor lymph, yet which carries nutrients to cells and waste products away from them. Thus the growth of erythemas does not follow preformed channels, and this fact explains the genesis of roundish disks, which develop in a manner not unlike that of cultures of bacteria and cocci growing on gelatin or agar plates.

In reality spots which extend evenly in all directions usually do not appear to be round, but rather oval-shaped. This observation is explained by the fact that in most regions the skin is subject to greater tension in one direction than

217) Grouped lesions with a denser group or a large "mother lesion" in the center surrounded by more or less loosely scattered satellites (from Latin *satelles* companion) (Fig 218) are termed *corymbiform* a word originally used to designate a cluster of flowers The corymbiform type of syphilid is also called *bomb-crater* syphilid which is a better comparison because its arrangement calls to the mind the effects of an exploded bomb which centrally has torn up a big crater that is surrounded by much smaller craters, which decrease



Fig 217 —Herpetiform arrangement (herpes simplex on arm)



Fig 218.—Corymbiform arrangement (verrucae vulgares on elbow)

in number toward the periphery Thus one might speak of a *bomb-explosion-like* grouping To explain the retarded growth of the satellites partial immunity in the vicinity of the original lesion has been assumed

The opposite of grouping is *dissemination* which means that the lesions are scattered over a large area Such disseminated skin eruptions are characterized by their distribution by the number and types of their elements and also by the *size* of the individual lesions. In a simple and for all practical purposes, sufficient manner the latter can be indicated by comparisons. The expressions

in another The *direction of tension* in various regions has been ascertained by piercing the skin of a cadaver with a round awl which upon withdrawal leaves not a round hole but an oblong slit. Therefore one speaks of the line of *cleavage* of the skin The ovals of skin lesions are oriented with their longest diameters in the directions of the lines of cleavage The ovalization so to speak of the lesions is well demonstrated in scabies (Fig 26) in tinea versicolor (Fig. 168) and most impressively in pityriasis rosea (Fig 183)

If peripherally extending lesions merge *polycyclic* figures result with convexities always directed outward in the direction of growth (Fig 219) If the original lesions and the resulting polycyclic areas are very small, as in grouped vesicles (herpes) the edges may be called *microcyclic* These configurations contrast with dendritic lesions, which have branchlike (dendritic) extensions and eruptions with jagged outlines comparable to geographic maps. The *dendritic* shape is encountered when peripheral extension follows lymphatic channels and also in very irregular bizarre fashion in keloids. *Geographic map patterns* are typical of some congenital malformations of the skin, especially pigmented and vascular nevi The geographic patterns may be divided into those with relatively straight borders (the pigmented spots of Von Recklinghausen's disease) and those with markedly jagged outlines reminiscent of peninsulas and islands (most simple pigmented nevi) Thus the "geographic maps" may be "closed" or may have extensions and stray lesions along their edges. If the outlines of the lesions appear particularly bizarre with straight lines rectangular corners, etc. one should always consider the possibility of an *artifact* (e.g berloque dermatitis from trickling drops of cologne) or the sequelae of some type of therapy (e.g irritation from adhesive tape or in the case of bullae medicated plasters or radiation with rectangular protective shielding)

Round patches which grow peripherally may fade sink or heal completely in the center changing a discoid or nummular eruption (Fig 219) into an annular (ring-shaped) one (Fig 222) *Circinate* literally means circle-shaped (*circos* Greek for "circle") but the term is frequently used for relatively small lesions which do not form complete circles but consist of arcs or segments of circles (Fig 223) It is not a well-defined or reliable term. The *centers of erythematous rings* may often exhibit a passive more livid hyperemia in contrast to the more bright red, actively hyperemic edges. In chronic cases the centers may be hyperpigmented depigmented (Fig 222) or atrophic They may also contain some residual elements of the original skin lesions or there may be recurrent lesions in the centers after transient healing Such central recurrences may spread peripherally and may heal again centrally to result in two or even more *concentric rings* (trichophytosis superficialis) It is characteristic of tinea imbricata that the number of scaly and concentric rings is particularly large If the eruptions which succeed one another in ring fashion are



FIG 219.—Confluence by centrifugal growth (melanodermatic arsenical erythema in left groin)



FIG 220.—Confluence by apposition of papules (lichenoid arsphenamine dermatitis)



FIG. 221.—Diffuse spreading (eczema erythematosum on buttocks)

By healing in some places and peripheral progression of remaining remnants interrupted rings (Fig 227) half rings, or kidney-shaped arrangements may develop (trichophytoid eczemas, psoriasis, tertiary syphilids, Fig 228) In such cases one can also refer to *gyrate* (twisted in a ring or spiral) *serpiginous* (creeping) *garland-like* or generally *figurated* eruptions. If the eruption has involved most parts of the body surface leaving only small islands of normal skin these



Fig 224 —Iris-type lesion, purely macular (erythema exudativum multiforme)



Fig 225 —Iris, composed of an erythematous edge, urticarial wheal, vesicle (erythema exudativum multiforme on the back of hand

latter of course have arciform edges but with *concavities* directed outward (Fig 229)

Many annular and arciform groups of lesions exhibit on their convex outward sides evidence of activity while on their concave sides they show signs of regression. This situation may also be reversed and despite progression outward the more active inflammation (e.g oozing with little crusts) may take place on the inner sides of the rings (Fig 230) This picture indicates that the

not entirely equal but have different colors or are composed of even different lesions one speaks of *iris* (rainbow) or *cockade formation* For instance this is true of *erythema exudativum multiforme* In this condition there may be a bright red outer zone surrounding a paler inner zone and finally a central lesion which again is dark red at its periphery and paler toward its center (Fig 224) Sometimes a red halo may surround an urticarial elevation which has a vesicle in its center (Fig 225)

Confluence of growing rings may result in the formation of figure-eight, clover leaf or similar more irregular arciform figures which are always composed of segments of circles the convexities of which are directed outward (Fig 222) Figure 226 *A–D* shows how such spreading and confluence take place



Fig. 222 — Annular configuration (psoriasis)

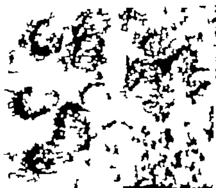


Fig. 223 — Circinary arrangement (eczema)



Fig. 227 —Interrupted ring (trichophytosis superficialis on the neck)



Fig. 228 —Kidney shape (tertiary syphilis)



Fig. 229 —Intact area of normal skin with concave edges (erythema annulare)



A



B



C



D

FIG. 226.—Centrifugal spreading with coalescence (erythema an ulare squamosum Siemens-Jagtman) *A* *D* *same skin area in consecutive stages of development.*

toward the peripheries. This may result from the decreasing concentration of the etiologic toxic agent with progressive diffusion from the centers of the lesions.

Rings and segments of rings may develop not only by peripheral growth of lesions but also by *apposition*. This is the case when new lesions continue to develop near the edges of old ones and, at the same time, regressive processes or complete healing takes place in the older more central lesions (Fig 233) Such rings which have formed by apposition are sometimes very small, hardly larger than lentils, and are called *gems* or *medallions* (Fig 234) The genesis of rings by apposition is also difficult to explain without assuming some immunization processes.

Finally ring and arc formations are occasionally seen in epitheliomas which grow in all directions but at the same time tend toward involution in the center (Fig 235) Arciform configurations, therefore may be of infectious toxic, or neoplastic origin.

Clearly different from the arciform types of configurations are the net-shaped or *reticular* arrangements encountered in passive hyperemias and their associated inflammations and pigmentations (Fig 16)

Despite their relative rarity *linear* shapes of skin diseases are of special interest. Streak-shaped lesions may obviously be of *exogenous* nature. This is the case for example, when drops of an irritating or sensitizing liquid run down the skin (berloque or locket dermatitis) or when stiff stems of grass scratch a sensitized skin (meadow dermatitis) It also occurs when a stripe-shaped area is exposed to pressure (melanoderma from suspenders) or when skin parasites make linear burrows (creeping eruption) In some disseminated dermatoses, a few linear lesions may develop in addition to the ordinary ones, and these usually follow pressure or scratch marks (provocation by irritation in purpura, psoriasis, lichen planus, verrucae Fig. 236) Still more remarkable however are those streak-shaped lesions which apparently develop without external causes. In some instances such as the pigmentation of the linea alba in pregnancy one can explain them on the basis of a preformed linear or stripe-shaped tissue arrangement But in other cases they pose a puzzle which we are unable to solve Morphea not rarely develops in the shape of a band in the center of the forehead (*coup de sabre* French for a blow with a sword") Great interest by dermatologists has been focused on linear congenital malformations or nevi. In the search for an explanation of such *endogenous* band or stripe formations some congenitally determined system has been postulated which—as in the case of the linea alba—is preformed in the skin and which the nevus only needs to follow in order to explain its linear shape. In view of this postulated existence of such an embryologically preformed system linear nevi have been called *systematized* nevi. Unfortunately however all efforts to discover this system have essentially failed. There are occasional linear-shaped

disease process needs a relatively long time to reach its climax from which it then regresses and finally heals. If *both* sides of the rings have signs of recent progression (as is occasionally encountered in psoriasis) we are dealing with recurrent eruptions in the territory of previous annular exanthems. The circinate growths of skin lesions with central clearing can be likened to the growth patterns of toadstools growing in forests in ever enlarging circles (Fig. 231) or of lichens forming rings on rocks and walls. *In a similar manner the growth of these skin lesions is halted where one circle touches the territory of another one as if the substrate had been exhausted.* Nevertheless we should by no means think of such ring and arc formations in terms of exhaustion of nutrients in the previously affected more central areas. We are dealing rather with the expres-



Fig. 230.—Acute symptoms (crusts and scales) along the inner edge of a ring (mycosis fungoides)

sion of some kind of local immunization. *This follows from the fact that not only* microörganisms (trichophytosis, syphilis, tuberculosis) but also toxic agents can give rise *to such figures (urticaria). In the latter case ring formation may* also be multiple or concentric (dermatitis herpetiformis Duhring, Fig. 232). The immunization or refractoriness in the centers of such rings *also* explains the extinguishing of rings at crossing points and the central recurrences. In the infectious diseases, recurrences also indicate that immunization was not complete and that virulent microbes have been able to survive in the partially immunized territory. These observations in diseases showing concentric ring formation permit the conclusion that in such cases local immunization runs its course in periodic waves. Toxic eruptions, however, may also show a reverse picture, with the most violent signs in the centers of the lesions and fading

toward the peripheries. This may result from the decreasing concentration of the etiologic toxic agent with progressive diffusion from the centers of the lesions.

Rings and segments of rings may develop not only by peripheral growth of lesions but also by *apposition*. This is the case when new lesions continue to develop near the edges of old ones and at the same time regressive processes or complete healing takes place in the older more central lesions (Fig 233) Such rings which have formed by apposition are sometimes very small hardly larger than lentils and are called *gems* or *medallions* (Fig 234) The genesis of rings by apposition is also difficult to explain without assuming some immunization processes.

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Fig. 231 — Mushrooms growing in a ring (after Kreutzer)



Fig. 232 — Concentric rings (dermatitis herpetiformis Duhring)



Fig. 233 — Large ring by apposition of lesions (trichophytoid papulo-squamous eczema)

discolorations or inflammatory erythemas and swellings which follow the course of blood and lymphatic vessels (thrombosis lymphangitis, sporotrichosis) No association however can be established between the lines of distribution of nevi and the courses of such vessels. It has been observed repeatedly that linear nevi follow the whorls of hair patterns or the lines where several such whorl areas adjoin. We also sometimes find in such nevi similar characteristic whorl formations (Fig 237) which apparently coincide with the hair whorls (Fig 238) This point is not easy to establish because the types and sites of hair whorls are

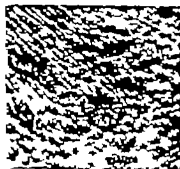


Fig. 234.—Small ring formed by apposition of lesions: medallion (lichen planus)



Fig. 235.—Ring formed by centrally ulcerating neoplasm (epithelioma spinocellulare)



Fig. 236.—Linear provocation by scratch ([illegible] planus)



FIG. 231 —Mushrooms growing in ring (after Kreutzer)



FIG 232 —Concentric rings (dermatitis herpet f rmis Duhring)



FIG. 233.—Large ring by apposition of lesions (trichophytoid papulo-squamous eczema)



Fig. 237.—Whorl formation in systematised naevus (naevus keratosus on trunk)



Fig. 238.—Hair whorl over angle of jaw

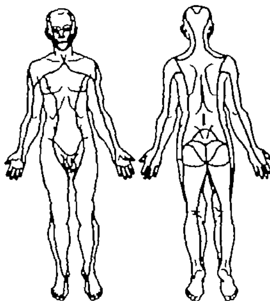


Fig. 239.—Voigt lines (border lines of areas of innervation by the cutaneous nerves)

subject to pronounced individual variation The localization of linear nevi in Voigt s lines i e the borders between *areas of innervation by cutaneous nerves* (Fig 239) is still more dubious since a rough congruence can be found only on the legs (Fig 240 *A B) but not on the arms or trunk.* On the legs such congruence when it occurs may be coincidental Such coincidence also seems the *more likely* because any congruence on the legs is not a perfect one (Fig 241)

In the majority of cases a coincidence of the nevus lines with any other known system of lines in the skin cannot be established If for instance, one examines more closely the lines of Blaschko i e the lines which have been arrived at as an empirical average from a large number of systematized nevi *(Fig 242) it will be found that out of the three characteristic peculiarities* of these lines only one—namely whorl formation—can be recognized in hair current distributions but that the other two characteristic features of these lines are lacking in all known phylogenetic systems of lines These other two special features are V shaped figures paramesially to the spine (Figs. 243 and 244) and S shaped arcs on the lateral abdomen and thorax *(Figs 245* and 246) which tend to be situated more medially in the lower and more laterally in the upper part *of a line which runs from the symphysis to the anterior axillary fold.* Therefore to explain the linear formation in nevi and other systematized dermatoses we are led to postulate the existence of an *autonomous line system* in the skin which becomes known to us only by the appearance of the dermatoses in question and which in its own way furnishes impressive evidence for *the independence of the skin from the other tissues and organs of the body* The patterns of the stripes and lines of animals are just as autonomous Therefore some have believed *that the nevus lines are analogous to animal designs* and that they represent an atavism i e the recurrence of a characteristic of some far remote ancestor This belief however is also not correct because in animal patterns the most striking peculiarities of the nevus lines (S- and V-figures whorl) are unknown.

The features of the *borders* of foci of skin diseases often deserve special attention Such borders may be well defined (trichophytoses and other mycoses psoriasis nevi) *or ill defined (Fig 247) The areas of disease may stop sharply* and definitely (erysipelas) or there may be stray lesions in the healthy surrounding skin (eczema Fig 14) The lesions may occur clearly on unchanged skin or they may be surrounded by border zones which are hyperemic or anemic or hyperpigmented or depigmented

The *distribution* of skin diseases is very interesting as well as puzzling The diagnostic importance of distribution has been much overrated in the past Even *today* the most frequent diagnostic errors of the beginner are due to a tendency to rely too much on the sites where eruptions occur instead of first closely studying the *nature of the lesions.* Thus lupus vulgaris of the face may be confused with lupus erythematosus and acne vulgaris with acne rosacea



Fig. 237.—Whorl formation in systematized nevus (nevus keratosus on trunk)



Fig. 238.—Hair whorl over angle of jaw

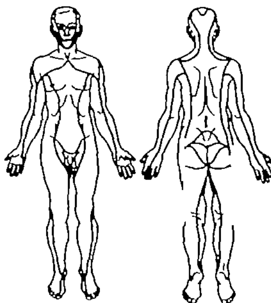


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FIG 242 —Blaschko lines (empirically established nevus lines)



FIG 243 —V shaped figure beside the spine in systematized nevus (after Blaschko)

A *B*

Fig. 240.—Linear eczema (*A*) and linear nevus (*B*) following Voigt's lines

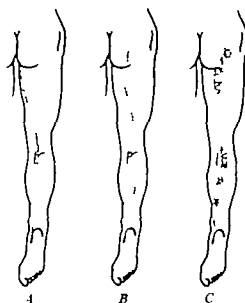


Fig. 241.—Deviations from Voigt lines *A* Voigt's line *B* linear eczema from Fig 240 *A* *C* linear nevus from Fig 240 *B*

places where they occur more frequently than elsewhere There are especially great differences in regard to extent of involvement of hairy and so-called non-hairy skin of extensor and flexor aspects, of covered and exposed areas (face neck V forearms) Relationship to exposed areas may be deceiving as in atopic eczema, for example which in men as well as women frequently occurs on the upper chest and shoulders (*en pèlerine*) and yet only in women is such



FIG 246 —S-shaped figure on the side of the chest (lichen planus)



FIG. 247 —Indistinct border (atypical psoriasis)

skin commonly exposed. In *different stages* of a disease, *different sites* may be predilectionally involved Thus secondary syphilis develops a disseminate exanthem over the trunk while recurrent eruptions have a marked tendency to be localized (anus, genitalia, face hairline oral mucosa) In variola which is spread quite evenly over the entire body the prodromal erythemas appear only in certain regions (femoral triangle) Frequently the involvement of mucosal surfaces is characteristic being common in some diseases (lichen planus

etc. Actually there is perhaps only one dermatosis which can be recognized with sufficient certainty from distribution alone and this is scabies (Fig 248) The places where itch mites settle are very typical indeed There is no other disease which appears in the interdigital spaces on the wrists about the navel along the axillary folds and on the nipples penis buttocks and edges of the feet and which at the same time leaves the back and head free. Despite its inadequacy alone the study of distribution may be an important diagnostic help All dermatoses have more or less pronounced *sites of predilection* i.e



Fig 244 —Left sid of a V shaped figure beside th spine (evus systematicus)



Fig 245.—S-shaped figure on the abdomen (nevus kerat ticus)

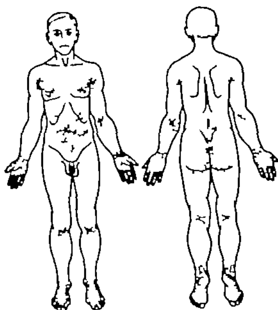


FIG. 248.—Distribution of scabies

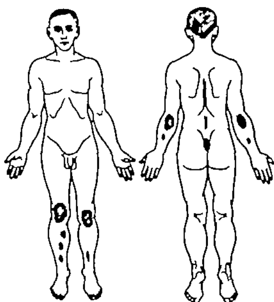


FIG. 249.—Most common areas of eruption of psoriasis

pemphigus) and practically non-existent in others (psoriasis) Thus knowledge of sites of predilection will frequently help guide the examination If there is a suspicion of psoriasis one will have a quick look at the elbows and knees the sacral area and the scalp (Fig 249) in lichen planus at the flexor aspects of the forearms and the oral mucosa and in vitiligo at the anus But one should always keep in mind that the distribution of a particular disease may occasionally be *reversed* (inverse) *with the eruption occurring just in those places* which are usually spared Thus we know a type of psoriasis with marked involvement of intertriginous sites (axillae submammary folds navel groin Fig 250)

The *causes* of the distribution of skin diseases are known only in some instances In dermatoses which have been caused by *external injurious agents* the *site is often very characteristic* and points clearly to the etiology (lipstick dermatitis of the lips mouthwash dermatitis around the mouth matchbox and garter dermatitis on the thighs solar eczema of the exposed skin ointment dermatitis of treated areas occupational dermatitis) In other instances it is not the disease itself but the sites where the lesions appear which depend on external irritation This occurs when lesions of a disseminate eruption develop in already irritated places (scratch wounds pressure points inflammations of other kinds, Fig 251 also Fig 362) It is known as Koebner s phenomenon or the isomorphic effect of irritation or more simply as *provocation by irritation* In a less strict sense skin changes which develop under special *anatomical conditions* such as in scars (Fig 252) on the linea alba or in areas surrounding moles (Fig 253) the nipples (Fig 254) or other natural body openings (Fig 255) might be considered in a similar category Thus one might view such areas as especially sensitive or as sites of lowered resistance But these conjectures *often fail to help our understanding because in other cases just the* 'less resistant areas (scars, body openings etc) are spared and would have to be considered desensitized or sites of increased resistance Only exceptionally can such a *localization be understood as in the case of papular syphilis* which spares cinnabar tattooed areas obviously because of the antitreponemal effect of the mercury deposits in the skin

Finally some special localizations are caused by the relations of some dermatoses to the *adnexa* Follicular diseases of course cannot be found in regions without follicles (palms and soles) Abscesses of the apocrine sweat glands (hydradenitis) occur only in the adult and exclusively in such areas as the axillae where such sweat glands are found

The relationships of skin diseases to the *blood vessels* are remarkably scanty As has been mentioned before it is true that the spots of roseolas and the meshes of cutis marmorata indicate regions of direct and indirect cutaneous blood supply However the supply areas of *larger* arteries as established by Manchot and Spalteholz fail to play a part even in the localization of vascular

a great and not very critical tendency to assume such connections. For example, *unilateral* distribution as occurs in the case of nevi was considered by some to be proof of an influence of the nervous system. This notion of course, is completely without foundation. *Bilaterality* and symmetry of a disseminate eruption is certainly often the expression of hematogenous dissemination of its causative agent. Bilaterality and symmetry however may also be rather common phenomena in other types of dermatoses. One should keep in mind that the skin itself is a bilaterally symmetrical organ. In fact, it is usually more



Fig. 252.—Vitiligo in scar (thigh, anterior aspect).



Fig. 253.—Vitiligo surrounding pigmented nevus (upper arm).



Fig. 254.—Psoriasis surrounding nipple.

nevi which also seems to be governed entirely by the skin itself Obviously the great number of vascular anastomoses leads to such a complete confluence of the capillary network of adjoining vessels that the areas supplied by individual arteries are not apparent as independent territories. Only in the case of embolisms of the popliteal arteries or of the arteries of the distal phalanges of the fingers may the ensuing proximal gangrenes be sharply bordered.

A definite relationship between *nerves* and the site of skin diseases can also only exceptionally be established In the early days of dermatology there was



Fig 250 —Psoriasis, inverse, involving armpits, infra-mammary folds, navel groins



Fig. 251 —Vitiligo surrounding a patch of verrucous tuberculosis. Right hand is free of tuberculosis as well as of vitiligo

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Fig 252 —Vitiligo in scar (thigh, anterior aspect)



Fig 253 —Vitiligo surrounding pig-mented nevus upper arm

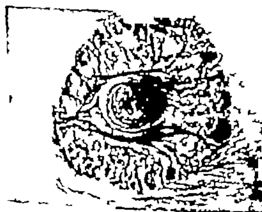


Fig 254 —Psoriasis surrounding the nipple

puzzling when an eruption is unilateral than when it is symmetrical This is especially the case when we see that even hematogenous exanthems, such as a syphilitic roseola can exceptionally appear on one side only a fact which so far has remained without explanation Unilateral distribution is well understood only if some external factor plays a role in the eruption e.g an exogenous infection (trichophytosis superficialis) In this case new lesions at first naturally form around the first focus of infection creating a unilateral eruption

There is one disease with a striking unilaterality namely *herpes zoster* The groups of vesicles which it produces not only are confined to one side of the body but are also limited above and below by the distribution areas of certain spinal

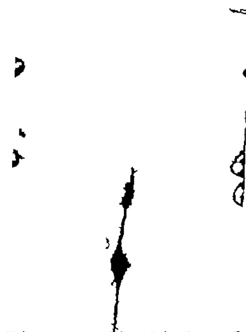


Fig 255 —Vitiligo perianal

nerves (Fig 256 *A* *B*) This distribution results from the fact that the disease is caused by an infectious inflammation of the posterior roots of the spinal nerves or more correctly of the spinal ganglia Therefore the spread of herpes zoster by no means corresponds to the area of a peripheral nerve (Fig 257) Some of the fibers from each ganglion run upward as well as downward to mingle with fibers of adjoining roots and therefore the herpes eruption extends over an area which is innervated by several neighboring nerves. Thus these segmental or metameric areas of innervation or radicular zones are *not identical* with the areas of ramification of the peripheral nerves. They are less sharply defined than the *latter*

Since the sensory nerves are accompanied by vegetative ones, some unilateral functional disorders such as hyperhidrosis unilateralis can be explained by the assumption of a unilateral nerve disturbance

A similarity in distribution to that of herpes zoster may be assumed by very dissimilar dermatoses if they happen to occur unilaterally In such cases the term ***zosteriform arrangement*** is used (Fig 258 *A B*) Of course, the borders, e g the median line of the trunk, are not so strictly respected as in herpes zoster Zosteriform arrangement is most frequently met in nevi a situation which formerly even led to the term "nerve nevi" even though these malformations

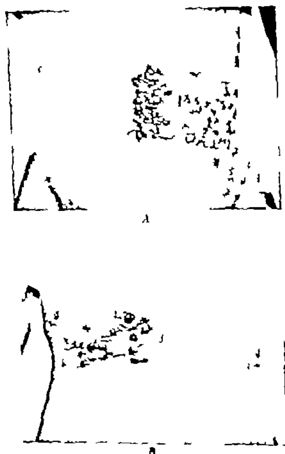


Fig 258—Unilateral eruption in the area supplied by a spinal nerve (herpes zoster) *A* anterior aspect *B* posterior aspect

have absolutely nothing to do with nerves They follow their own autonomous laws, as has already been discussed. They share their almost exclusive unilaterality with many other congenital malformations. Like all typically unilateral malformations, the unilateral nevi also, as a rule are not hereditary Unilaterality and absence of heredity are correlated phenomena. The cause of these remarkable skin changes and especially the genesis of their distribution are completely unknown.

Fig. 257.—Areas supplied by spinal nerves (after F. von Muller)



A



B

Fig. 258.—Zosteriform psoriasis mostly healed with hyperpigmentation. A, anterior aspect; B, posterior aspect.

In many other respects, too we do not know what causes the particular localization of skin diseases. In general except for externally caused dermatoses, this field is almost entirely dark and even mysterious. We do not even understand why one species of lice exclusively favors the scalp another the clothes and a third the pubic and axillary hair Nor do we know why the mite which causes scabies likes the abdomen and the wrists but not the back and the face It is not true that the skin in the different places where the mite likes to settle is particularly thin or thick or loose or adherent. It is an unsolved puzzle why the skin of the buttocks is almost always involved, while the skin between and over the shoulder blades remains free. Why also in addition to the axillary folds and wrists, are the *extensor* but not the *flexural* aspects of the elbows affected Why are the female nipples so much more often involved than those of the male while in the male the genitalia are so much more frequently affected than in the female? What makes the interdigital and axillary folds so susceptible while the folds in the face remain free Why do the anterior axillary folds so often and the posterior axillary folds so seldom participate in the eruption The tastes of these mites seem completely independent and unexplainable Just as specialized are the tastes of other pathogenic microorganisms on the skin such as the yeast fungi, which cause erosio interdigitalis just between the third and fourth fingers while the other interdigital spaces are only rarely affected. In some cases the localizations of foci of infection even change with the age of the host. For example the microsporon fungi involve the scalp of children most stubbornly but disappear spontaneously as soon as the children reach puberty On the other hand the fungus causing favus though it has the same site of predilection remains unaffected after puberty even though adults only rarely become newly infected. In older children and adults, staphylococci exhibit a marked predilection for the follicles (furunculosis) and affect the *sweat* glands only in the axilla while in small children they spare the follicles and are exclusively interested in sweat glands and their ducts (sweat gland abscesses and porites)

Also in *non-infectious ailments* we usually fail to establish more tangible facts than the predilections of certain skin diseases for certain body areas and as soon as we try to explain these predilections, we get involved in contradictions. Thus one is generally inclined to blame the greater delicacy of skin on flexor aspects for the localization of an eruption in these regions *But at least* as many skin eruptions favor extensor surfaces in spite of the undoubted fact that such skin is tougher and in all mammals better equipped with protective devices (pigmentation hair growth horn shields) This is even true in the case of the photodermatoses, where the more exposed and easier tanning skin of the extensor aspects is actually but unexpectedly more light-sensitive than is the skin on flexural aspects In any case the skin in different areas has differences other than those we can observe with our senses and our usual methods of

examination Generally symmetric areas correspond in their reactions, which explains why so many dermatoses which do spread by contiguity i.e. not in a creeping manner jump over to the other side (Fig 259 *A* *B*) The "jumping of a skin disease however takes place not only from side to side but also in "vertical correspondence as from the armpits to the groins (intertriginous eczemas epidermophytoses) from the eyelids to the genitals (eczemas) from



A



B

Fig 259 — *A* Vesicular irritation after treatment with 10 per cent pyrogallic acid in petrolatum in left groin Right groin, which had been treated blandly is free *B* jumping of the left-side irritation to the blandly treated right side a few days later

the feet to the hands (trichophytoses, dyshidrotic eczemas) or from the thighs to the trunk and arms (garter eczemas) which in the latter case may mark the beginning of so-called generalization of the eruption Therefore laymen often think even in eczemas in terms of a direct transfer of disease germs. In reality this jumping and for that matter also the simultaneous appearance of eruptions in different regions are usually only the expression of a functional similar

ity or inherent relationship between the respective areas of the skin. On the other hand, any area of the skin may at times exhibit a quite specific and lasting disposition toward disease, while the ordinarily corresponding sites remain free. This is well illustrated by fixed drug eruptions and the many circumscribed chronic eczemas. These phenomena deprive us of even the last point by which to understand the many problems of localization and leave us with the necessity of vaguely assuming wholly individual and wholly localized disease predispositions of unknown origin. In this field it remains the privilege of the layman to believe that he knows *more* than the expert in the field.

While the beginner is rashly inclined to touch with his fingers the skin changes which he sees, the experienced dermatologist will first try to absorb the visible changes by quiet and thoughtful inspection. This of course must be followed by *palpation* which will uncover peculiarities of the disease process which remain hidden to the eye.

Palpation can and must be executed in various fashions. By running the fingertip over the lesion, insignificant differences in level as well as in smoothness or roughness of the surface may be noted By touching with the dorsal aspects of the fingers, differences in temperature may be established. Controlled pressure on the changed skin may disclose its consistency or in other words, its softness or hardness. The *palpation of the borders* of a lesion may show its extent toward the depth. Frequently subcutaneous tumors and infiltrates can be found only by palpation (erythema induratum of Bazin) The *lifting of folds* provides information on the pliability of the skin in general, as well as its thickness and elasticity or capacity for expansion. Together with the lifting of folds, *shifting* the skin about permits us to determine the degree of mobility over underlying structures and also the connection of deeper lesions with the cutis and epidermis, on the one hand as against neighboring muscles and bones, on the other By vigorous stroking with the knuckles or with a blunt instrument or by friction, we can establish mechanical irritability (urticaria factitia urticaria pigmentosa) or the readiness to form blisters (pemphigus, epidermolysis bullosa) and, by blunt or rotating pressure by artificial stasis, the tendency to form cutaneous hemorrhages (purpura factitia) And, finally by scratching with one fingernail or with a little semiblunt curette (*grattage méthodique*) we can demonstrate latent desquamation examine the degree of adherence of horny deposits, and establish the tendency to bleed readily (psoriasis)

Palpation sometimes discloses surface peculiarities and especially *differences in level* including the smallest papular elevations, which are sometimes too small to be readily visible The sense *of touch also* can protect us from the optical delusion which makes bluish macules look like slight depressions. Fine desquamation is sometimes better *felt as* a roughness than *seen* (erythematosquamous eczemas) The grater-like feeling which one perceives by running a finger over closely set follicular keratoses is very characteristic (lichen ruber acu

minatus keratosis *follicularis spinulosa*) Conversely in other cases stroking makes one aware of a peculiar smoothness and softness (cutis elastica alopecia *universalis*) Dryness moisture content and greasiness of the skin surface may also be examined by the sense of touch

In some skin diseases the sense of touch informs us about *deviations of temperature* e g increased temperature in active inflammations and some large hemangiomas, decreased temperature in anemia and stasis (Raynaud s disease, scleroderma)

Much more important is the use of palpation for appraising *consistency* In the first place this applies to the consistency of the *skin in general* which of course differs in various regions and also changes with the general condition of the patient. It is dependent on many factors such as the thickness of the horny layer (palms and soles) the water content of the skin (turgor) the skin tension or tone (increased in cold decreased in old age) the relative amounts and conditions of collagenous and elastic fibers the density of the connective tissue reticulum the development of the panniculus adiposus and the tightness of the bond between cutis and subcutis or the underlying organs (lids genitals, and dorsa of hands as compared with palms and soles) The elasticity expansivity and pliability of the skin may therefore fluctuate within wide limits. The expansivity of the skin may be much increased in cutis elastica giving it at the same time a peculiar texture of softness, like suede leather A somewhat similar feeling is elicited by touching the completely hairless skin in alopecia universalis In cutis gyrata the skin forms gross movable folds. In cutis laxa non elastic pendulous empty bags of skin may be present In scleroderma elasticity and pliability may be completely abolished

Even more important is examination of the *consistency of certain foci of disease* Inflammatory states not only tend to make the skin less expansible and elastic and therefore less pliable but also cause it to appear tougher This is true of superficial (lichenification) as well as deep (furuncle) inflammations. There are also other types of cellular infiltrates which can be detected by palpation Very small papules of normal skin color may be perceptible by palpation only Small vesicles are also easily palpable because the pressure of their contents makes them very firm The papules of secondary and tertiary syphilis are conspicuous by resistant infiltration while tuberculous foci of inflammation are frequently softer than normal skin and feel mushy The floor of a syphilitic primary lesion appears very firm to the touch so that it is customary to speak of it as demonstrating induration or sclerosis (hardening) The carcinomas are often still harder It is therefore often possible by palpation to recognize with great certainty the neoplastic character even of the thin marginal extensions which surround ulcerated epitheliomas Also hard to the touch are of course all thickenings of the horny layer (calluses corns warts) as well as small crusts which have a further special quality of being sharp and brittle like glass For

this reason, very small punctate crusts are better diagnosed by touch than by the eye. The skin in scleroderma is as hard as a board and feels as if it were frozen while simple connective tissue condensations (elephantiasis keloids) are very tough, yet not hard. Accumulations of mucin (myxedema) are softer and even softer are cellular richly vascularized infiltrates particularly tissues which have been loosened by edema. It is also characteristic of edema that pressure with a finger leaves a hollow which is only slowly filled out. Cavities filled with a thick pulp (sebaceous cyst) may have a plastic quality and retain the molded shape given by finger pressure for a long time (Fig 260) If cavities are not too tautly filled with liquid (cysts, abscesses) they may exhibit the sign of *fluctuation* The palpating finger may feel such fluctuation if while one index finger palpates, the other index finger applies a short, jolting pressure to the lesion. A similar less



Fig 260—Doughy consistency of cyst with pulpy contents, (*A*) before and (*B*) after squeezing with fingers

distinct phenomenon, which is called *pseudo-fluctuation* may be observed in soft elastic structures such as muscle and fat tissue. Skin which is lacking elastic fibers (striae gravidarum) gives a very characteristic feeling of softness. This condition called *anetoderma* has already been described

In small pinhead-sized lesions which are not raised above the level of the skin the consistency of course cannot be tested with the finger In this case we can use a blunt *probe* to test how the abnormal skin responds to pressure with the instrument. This is especially useful in the diagnosis of lupus vulgaris. Because of its thin covering with epidermis and abundance of cells, the macule like lupus nodule is soft and mushy so that the probe breaks easily into the tissue and after withdrawal, brings a drop of blood. For definite diagnosis of vesicles and pustules, it is sometimes necessary to prick them with a needle or pointed scalpel to make sure whether clear fluid or pus emerges.

The most important purpose of palpation however is to provide information on the *extension of changes into the depth* and also on invisible processes

present below the surface Thus there are cutaneous-subcutaneous and purely subcutaneous nodules whose presence can be recognized only by palpation. Even larger nodules may sometimes protrude only so little above skin level that they are invisible This is particularly true of deep subcutaneous nodes, since a bulge becomes flatter and its edges less sharp *the deeper the causative mass of* condensed tissue is situated (erythema nodosum erythema induratum of Bazin) Abscesses and adenopathies also can frequently be diagnosed only by palpation In other cases the presence of a skin change is readily visible but only the palpating finger can establish the extent of the infiltrate or tumor in breadth as well as depth (furuncle tertiary syphilis, nodular tuberculid) It is frequently astonishing how far a palpable infiltrate which is covered by normal skin extends beyond its visible borders. This situation is called *submerging* of the infiltrate or tumor Together with the existence and size of such deep-seated nodules their shapes can be determined Cysts are characteristically ball-shaped lipomas are lobulated and carcinomas uneven or bumpy

Palpation in depth not only provides information about size and shape but also about *movability* and with the latter about the relationship of the mass to the surrounding structures Above strictly subcutaneous nodules and nodes, the skin is freely movable while cutaneous and cutaneous-subcutaneous infiltrates follow the movements of the skin Infiltrates and tumors, abscesses, scars and other masses which penetrate to greater depth and become firmly attached to the underlying tendons muscles or bones can no longer be shifted on their bases

CHAPTER FOUR

Hair and Nails

Hairs and nails are bloodless and insensitive horny structures which, because of their nature, are unable to respond to pathogenic causes in the same manner as the living integument does. When one examines *hairs* one has to keep in mind that the hairs of different regions of the body are very differently developed and that the different types of hair on the same person may react to a disease in different ways. Therefore in a hair disease one should not fail to inspect all hairy regions, one after another. If the patient complains about the scalp only, one should also examine closely the eyebrows and lashes, the beard, the axillary and pubic hair, and the lanugo on the trunk and extremities. Examination of the lanugo should be done under changing angles of illumination, which cause small glossy hairs to light up, or in tangential (skimming) light in front of a dark background, e.g., a dark corner. During examination of hairs, one should also try to visualize the possible changes, one after the other, so that nothing important may be overlooked. The changes to be looked for are *changes in color* of the hairs, *increased amount* of hair, *decreased amount or absence* of hair, *changes in shape* of the individual hairs, *deposits* on the hairs, such as (*a*) deposits produced by the body (scales, crusts), (*b*) *deposits* of foreign bodies (nits, fungi, matted bacteria, and fungous masses), *distinction and extent* of the hair changes.

COLOR OF THE HAIR

The color of the hair, which shows well-known individual and racial variations, may also change during the course of life (darkening after infancy and in childhood, bleaching by light, graying with age, discoloration by drugs). Hair color locally may also become darker (melanotrichia) or lighter (leukotrichia, canities) than other hair in a circumscribed area without affecting the skin. In other cases the discoloration may also be associated with changes in the skin, such as hyperpigmentation (nevus pigmentosus, Fig. 261, hyperpigmentation from light with melanotrichosis), thickening (nevus pellinus, fur nevus), or depigmentation (vitiligo, circumscribed, Fig. 262). In other cases, the skin may be erythemato-squamous (discolored hairs in dermatitis, e.g., trichophytosis) or the color change may be associated with the loss and regrowth of the hair (white hair in alopecia areata). Pili annulati (ringed hair) will be discussed later.

present below the surface Thus there are cutaneous-subcutaneous and purely subcutaneous nodules whose presence can be recognized only by palpation. Even larger nodules may sometimes protrude only so little above skin level that they are invisible This is particularly true of deep subcutaneous nodes, since a bulge becomes flatter and its edges less sharp the deeper the causative mass of condensed tissue is situated (erythema nodosum erythema induratum of Bazin) Abscesses and adenopathies also can frequently be diagnosed only by palpation In other cases the presence of a skin change is readily visible but *only the palpating finger can establish the extent of the infiltrate* or tumor in breadth as well as depth (furuncle tertiary syphilis, nodular tuberculid) It is frequently astonishing how far a palpable infiltrate which is covered by normal skin extends beyond its visible borders This situation is called *submerging* of the infiltrate or tumor Together with the existence and size of such deep-seated nodules their shapes can be determined Cysts are characteristically ball shaped lipomas are lobulated and carcinomas uneven or bumpy

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chosis sacralis lanuginosa, Fig. 264 hypertrichosis sacralis terminalis) or they may be associated with a variety of *skin changes* These changes may be permanent, such as the pigmentation and thickening of the skin in the furry nevus (Fig 265) or they may be transient, like the inflammatory phenomena (pyodermas, arthritides) which occasionally lead to irritative hypertrichosis, or the pressure and friction erythema which may elicit the hypertrichosis found on the shoulders of bag carriers

DECREASED HAIR GROWTH

Decrease in hair amount may come about by neurotic *pulling out* by *rubbing off* (as in the occipital area of infants and in itching eczemas) or by *breaking off* The latter may be caused by fungi entering the hairs (microsporons trichophytons) or it may be due to changes in shape of the individual hairs, which will be discussed later (trichorrhexis, monilethrix etc.)

Too scanty hair growth may be caused by *silky thin* hair associated with failure of lanugo hair to become thicker in later life (agenesis Fig. 266) In such cases the number of hairs may be normal or decreased On the other hand the hairs in scanty growth may be of more or less normal caliber but deficient in numbers (hypotrichosis, Fig 267)

Hair of originally normal density may become sparser after local inflammations (erysipelas, pityriasis capitis, eczema, Fig 160) after systemic disturbances (fever difficult childbirth, loss of hormonal equilibrium) or even from completely unknown causes (defluvium) If the shedding of hair is very marked it may lead to complete baldness (alopecia) which may also be a congenital condition. Alopecia may be circumscribed (Fig 268) it may involve the entire scalp (total, Fig 269) or even the entire body surface (universal, Fig 270)

When hairs are absent, the skin may be normal or pathologically changed. One first has to establish whether the follicles are still preserved. If so they can be seen as fine points or pits, as, for example in alopecia areata (Fig 271) If the follicles have disappeared, atrophy is present and the skin is smooth, glossy and lacks surface relief (Fig 272) (alopecia atrophicans, alopecia senilis, alopecia favica, lupus erythematosus) When such atrophic skin is pinched together between thumb and index finger it exhibits the characteristic pattern of little folds which is traditionally compared with crushed cigarette paper (Fig 273 *A B*) If the skin is hypotonic (meaning reduced in fulness and turgor without serious anatomic changes such as occur in scars) similar small folds may occur and look like atrophy (Fig 274) In this case, however the follicle mouths are still visible. Atrophic skin may also simultaneously manifest depigmentation, hyperpigmentation and telangiectases (traumatic scars, scleroderma, X-ray atrophy Fig 275)

In alopecic areas the skin may also exhibit features of inflammation, especially erythema (alopecia toxica) or erythema with desquamation (Fig 276) The

INCREASED HAIR GROWTH—HYPERTRICHOSIS

Increase in hair growth may affect the fine silky *lanugo* or the somewhat stronger *terminal hairs* (Fig 263) The lanugo may grow enormously with simultaneous failure to develop terminal hairs. This is true in the so-called monkey men (lanuginosis hereditaria) In these cases it is difficult to decide whether the condition should be called a *hypertrichosis* (viz lanuginosa) or a *hypotrichosis* (viz terminalis) It actually is a *hypertrichosis hypogenita* a hypertrichosis due to *insufficient* hair development

Hypertrichoses may also occur without demonstrable changes in the underlying skin, in generalized (see above) as well as in circumscribed form (hypertri-



Fig 261 —Hyperpigmented hai on hyperpigmented skin (nevu pigmentoso-pilosus on the scalp)



Fig. 262.—Depigmented hair on depigmented ski White forelock (albinismus areatus)

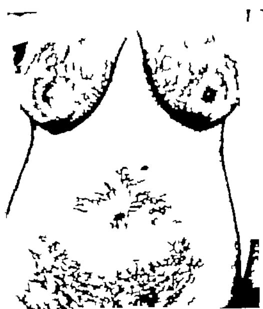


Fig. 263 —Hypertrichosis terminalis universalis

chosis sacralis lanuginosa, Fig 264 hypertrichosis sacralis terminalis) or they may be associated with a variety of *skin changes* These changes may be permanent, such as the pigmentation and thickening of the skin in the furry nevus (Fig. 265) or they may be transient, like the inflammatory phenomena (pyodermas, arthritides) which occasionally lead to irritative hypertrichosis, or the pressure and friction erythema which may elicit the hypertrichosis found on the shoulders of bag carriers.

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desquamation may spread diffusely over a large part of the scalp as it commonly occurs in the early stages of seborrheic dermatitis associated with common baldness, or the scales may be found at the bases of the still existing hairs (alopecia atrophicans) The lost hairs may also be replaced by keratinous spines (keratosis follicularis spinulosa decalvans Siemens) or by plugs (lupus erythematosus Fig 277) which sometimes may even be of very large circumference (keratosis follicularis acneiformis)



Fig 264 —Hypertrichosis lanuginosa circumscripta sacralis congenita.



Fig. 265 —Hypertrichosis on pathologically changed skin (furry nevus on right cheek)

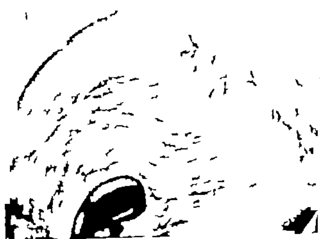


Fig 266.—Too thin and too few hairs (genesis)

CHANGES IN SHAPE OF INDIVIDUAL HAIRS

The examining physician also must pay attention to *changes in shape of individual hairs* and to the phenomena which are caused by these changes. Elliptic, instead of round cross-section causes *curly* hair Abnormal keratinization of the skin at the mouth of the follicle may prevent emergence of the growing hair from the follicle and force it to roll itself into a spiral beneath the surface and cause an inflammatory nodule containing an *ingrown hair* Sometimes ingrown hairs are visible through the transparent horny layer (Fig 278) as little rings (rolled

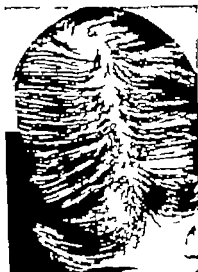


Fig 267 —Too few hairs (hypotrichosis hereditaria)



Fig. 268 —Circumscribed alopecia (alopecia triangularis in pseudo-hermaphroditism)



Fig. 269 —Total alopecia (alopecia areata of the scalp)

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Fig. 264 —Hypertrichosis lanuginosa circumscripta sacralis congenita.



Fig. 265 —Hypertrichosis on pathologically changed skin (furry nevus on right cheek)



Fig. 266.—Too thin and too few hairs (agenesis)



A



B

FIG 273 — *A* alopecia with atrophic wrinkling (lupus erythematosus); *B*, healed



FIG 274 — Alopecia with hypotonic (pseudo-atrophic) wrinkling The follicle openings are visible (alopecia areata)

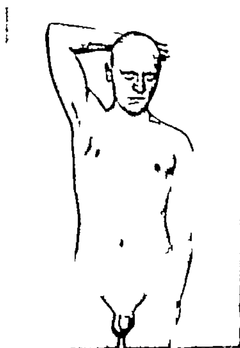


Fig 270.—U lversal alopecia (alopecia areata uni 'ersalis)



Fig. 271 —Alopecia with visil le follicle openings (alopecia areata)



Fig 272 —Alopecia and atrophy \ te bsence of f llicles (alopecia atrophicans)

hair) Peculiar phenomena are caused by *splitting* of the hair either at the end (trichoschisis) or within the shaft (trichorrhexis Fig 279) which may appear as a little gray *nodule* or often a *kink* at the site of the split. In other cases the impression of a little nodule in the hair shaft may be created by actual rolling of the hair into knots (trichonodosis, Fig 279) which may cause the hair to fray and break off A periodic knoblike thickening of the hair may also be simulated by *twisting of the hair around its longitudinal axis* (trichokinesis or pili torti Fig 279) In this anomaly chiefly the very thin hairs are slightly curled and have a peculiar mottled luster due to the altered reflection of light from the twisted hair shafts (Fig 280) *Intermittent apparent swellings* which actually result from periodic thinnings of the hair shaft (Fig 279) are found in monilethrix. In this condition the hairs almost regularly break off near the base at one of the thinnings, so that the condition gives the impression of alopecia. Another



Fig 278 —Rolled hairs

condition which may present the appearance of swellings of the hair shaft arranged in beadlike fashion may on closer examination, turn out to be simply white or paler air-containing sections alternating with dark and air free portions (pili annulati, ringlet hair Fig 279) The dark parts of the hair give the impression of beads. Most of these changes can, of course be differentiated only with a magnifying glass or microscope.

DEPOSITS

Deposits on the hairs may be foreign or derived from the body The latter are scales and crusts. *Scales* may be dustlike small, or larger particles adhering loosely to the hairs, or they may partly sheathe the length of the shaft (tinea amiantacea, psoriasis Fig 175) In the latter case the hairs may be matted together forming hair tufts which, in psoriasis, are frequently arranged in rings (Fig 281) Of course *crusts* can cause hairs to stick together even better (Fig 282) and with sufficient neglect, there may even form an inextricable mass of hair serum pus, and lice Such impetiginized types of the pediculosis



FIG. 275.—Alopecia with poikilodermatic and atrophic skin



FIG. 276.—Alopecia toxic, with central erythema and scales (trichophytosis profunda)



FIG. 277.—Alopecia due to replacement of hairs by horny plugs (lupus erythematosus)

EXTENT OF HAIR CHANGES

Finally in every case of hair disease, we must consider the *distribution and extent* of the morbid process. A hair disorder may occur in a circumscribed focus or may be generalized, affecting many hairy areas (hypertrichosis generalisata) Finally such hair diseases may be universal involving the entire body surface (alopecia universalis, Fig. 270) A hair disease may affect all hairs or only medullated hairs (defluvium after typhoid fever) In other cases the hair changes are *regional* involving only certain areas, such as the scalp bearded area, axillary region, etc.



Fig 280 —Thin hair and mottled luster in trichokinesis (pili torti)

Of the greatest practical importance of course are the diseases of the *scalp* hair Here again there are diseases which are *circumscribed* occurring in a confined site (leukotrichia circumscripta) *areated* affecting several areas (alopecia areata) or *diffuse* without sharp borders and covering a large part of the scalp (alopecia prematura on the vertex, hypotrichosis hereditaria, defluvium) If the scalp hair is completely affected, the condition is called *total* (alopecia totalis) which differs from universal (see above) If there are isolated foci, one must distinguish between diseases characterized by *large lesions* (alopecia areata) and those with *small lesions* (alopecia syphilitica alopecia parvimaculata or parvi areata)

Pathologic phenomena in the *nails* are dominated by the fact that a dead disk of horn in the case of disease is able to produce only a much smaller variety of changes than the living skin. Essentially only discoloration, irregularities of

capitis (trichoma plica polonica) once played an important part in dermatology

Among *deposits of foreign origin* is the light-colored *flourlike coating* with which the microsporon fungi cover the hair Nits, as the eggs and eggshells of head and pubic lice are called are light-colored glossy *discrete nodules* which are attached to the hair like little buds (Figs 283 and 284) Dark, very hard

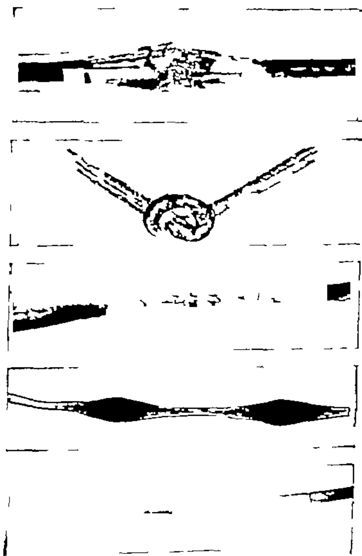


FIG. 279 —Trichorrhexis, trichonodosis, trich linearis, monilethrichia, pili annulati

granules of otherwise similar appearance containing fungus spores, are found in tropical piedra while in the related European trichomycosis palmellina there are granules or longer irregular sheaths of yellow to red color surrounding the hairs (Figs 285 and 286) These granules and sheaths consist of fungi and cocci which are imbedded in a gluey mass (zoogloea)

the surface brittleness, thickening and thinning and finally detachment from the nail bed are possible Therefore, the diagnosis of a disease frequently cannot be made from the symptoms in the nails alone For example it is often not possible to distinguish clinically with sufficient clarity eczema and psoriasis of the nails from the onychomycoses. In this case a decision about the diagnosis rests on the examination for fungi Only occasionally is a certain phenomenon in the nails *diagnostic* of a skin disease (pitted nails of psoriasis) The general rule never to consider a dermatological diagnosis as definitive without having examined the entire skin surface of the patient is particularly useful in the case of obscure nail diseases By so doing the physician may occasionally succeed in establishing an apparently impossible diagnosis. This is well illustrated by the case of a schoolgirl with a chronic hyperkeratosis on the thumb the psoriatic nature of which could be assumed when a single, only lentil-sized yet typical, psoriasiform scaly lesion in the sacral area was discovered (Figs. 287 and 288)

When *examining the nails* one should proceed just as methodically as in the examination of the skin and hair If one fails to do so one will encounter great difficulty in analyzing the features of an unusual picture crowded into a small field Therefore, one should systematically consider one after the other the following details *shape* of the nail plate *color* and *translucency* condition of the *surface* (level differences) *scaliness splintering crumbling thickening thinning* or even complete absence of the nail plate *localization* and *extent* of the nail change *detachment of the nail consistency* (hardness and softness) of the nail plate (palpation findings) and changes in the *surroundings of the nail* (nail wall cuticle)

CHANGES IN SHAPE

The nails may be abnormally large or abnormally small. *Enlargement may* occur together with enlargement of the *whole finger* (megalonychosis in acromegaly) or it may be restricted to the *tip of the finger* In the latter case the nail may *bulge* like a balloon and be excessively curved longitudinally (Hippocratic curving of the nails) This shape occurs in the so-called *clubbed fingers* which develop as a result of heart and lung ailments (especially bronchiectases) but which may also occur independently of such ailments (Fig. 289)

The nail may also be unusually *shortened* This may be a hereditary anomaly (especially on the thumbs) it may be the result of nail biting or it may be associated with a vascular nevus In the latter case the whole finger is thickened, and the nail is also enlarged in width (Fig. 290)

Of course the nail plate may be of normal size but otherwise *deformed* An example is the familial anomaly called double-edged nails. Here the nails have a plateau like cross-section with a sudden steep slope on both sides instead of the usual gradual transversal curvature. In some nails the normal gentle longitudinal curvature is absent which results in *flat* horny disks (platyonychia)



Fig 281 —Bundles of hair held together by scales (psoriasis capitis)



Fig 282 —Bundles of hair held together by crusts (pediculosis capitis with impetigo)



Fig. 283 —Nits (pediculosis capitis)



Fig 284 —Nits, enlarged

Fig 293) Single white transverse bands of about 1 mm in width may form in all nails at the bases during fever bouts or poisoning (arsenic) and then travel outward in a distal direction with the nail growth (Reil's lines) Together with other nail changes, such as thickening longitudinal grooves, and brittleness, leukonychia occurs in more or less *longitudinal stripes* in trichophytosis and in favus (Fig 294) Here too, the whitening may become a total one. A *white silvery luster* may come about if desquamation or splitting of the nail permits air



Fig 288 —Solitary typical psoriatic lesion in the sacral area Same patient as Fig 287

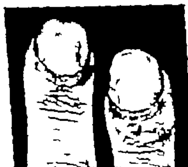


Fig 289 —Enlargement of nails Clubbed fingers in pneumothorax

to invade the nail substance there causing reflection of light much in the same way as happens in the horny layer of the skin. Thickened nail plates are usually *turbid and yellowish or brownish to blackish* (phaeonychia) the same colors as the color of horny substance elsewhere (phaeoderma) Exceptionally a nail or part of it may be *dark brown from melanin pigmentation* (melanonychia, Fig 295) Blood blisters under the nails appear dark red to black. A variety of discolorations may be caused by subungual tumors (angiomas, melanomas, endotheliomas, verrucae etc.)

Following injury pathological keratinization or subungual keratoses the nails may appear lifted up like the keel of a ship The nail plate may also be *slanted* as has been observed on the index fingers in cases of hydroa aestivale and epidermolysis bullosa (Fig 291)



Fig. 285 —Trichomycosis palmellina



Fig 286.—Trichomycosis palmellina enlarged



Fig. 287 —Thickening of the thumbnail in an otherwise apparently healthy girl

DISCOLORATION

Frequently the nail plate exhibits little *white spots* (leukonychia punctata good luck spots) which are apparently caused by air penetration They have a tendency to line up in *transverse bands* (leukonychia striata Fig 292) Sometimes, of course, the *entire* nail plate may appear white (leukonychia totalis,

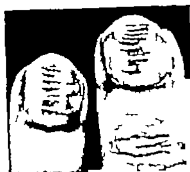


Fig 292.—Transverse white lines (leukonychia striata)



Fig 293.—Total whitening (leukonychia totalis)



Fig 294.—Longitudinal white bands (trichophytosis)



Fig 295.—Longitudinal brown band (melanonychia)



Fig 296 Discoloration by drug Trypaflavin)



Fig 297.—Polished surface (glossy nails in generalised eczema)

Of course a striking discoloration of the nails may be caused by foreign dyes which get into contact with the nails either occupationally or as medicaments (Fig 296) These discolorations travel distally with the growing nail

IRREGULARITIES OF THE SURFACE

The surface of the nail may be either *smoother* than normal or *less smooth* and show surface irregularities The former condition glossy nails which makes

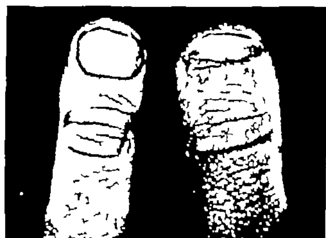


Fig 290 —Shortening of a nail (In nevus flammeus)



Fig 291 —Oblique position of th nail of the index (epidermolysis bullosa simplex tarda)

the nails look as if they were polished (Fig 297) is encountered in persons with chronic itching dermatoses who rub their nails continually against the itching skin.

Longitudinal ridges of the nails may occur in the senile (Fig 298) They may also be associated with *grooves* (e g after injury Fig 299) or the grooves may be especially marked (e g after injuries or associated with fibromas of the nail



Fig. 298.—Longitudinal ridges (senile nails)



Fig. 299.—Longitudinal ridge and groove (due to a scar in the root of the nail)

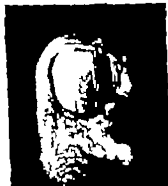


Fig. 300.—Longitudinal grooves, due to fibroma in the nail wall in tuberous sclerosis



Fig. 301.—Longitudinal grooves with splintering (onychodystrophy)



Fig. 302.—Longitudinal and transverse grooves (onychogryposis)

wall in tuberous sclerosis of the brain Fig 300) Frequently associated with ridges and grooves are desquamation and splintering (fraying) as in onychodystrophy (Fig 301) Transverse ridges are common in onychogryposis (Fig 302)

More frequently *depressions* are encountered in the nail plate. Most remarkable are the numerous small sharp-edged pits which are diagnostic of psoriasis (pitted nails Fig 303) Sometimes these pits are arranged in longitudinal rows (Fig 304) Sometimes even relatively few typical pits are sufficient to establish the diagnosis (Fig 305) It is however true that similar pits also occur in eczemas and other nail diseases, but then they are usually not so regular or so sharply punched out Besides such small pits the nail surface may exhibit larger trough shaped less sharp sometimes predominantly longitudinal but *more frequently transverse irregularities of the surface* (especially in eczemas and dermatomycoses, Fig 306) There may also occur an evenly *flat* depression of the entire nail which because of its appearance has given rise to the term *spoon nail* (koilonychia Fig 307) In this condition even the whole fingertip may be distorted as if it had been pulled up in a dorsal direction (Fig 308) Transverse furrows which run across all nails (sulci transversales) and are sharply demarcated are usually called *Beau s lines* They may consist of simple *transverse grooves* sometimes with collarette like scaly edges (Fig 309) or they may also cut transversely through the entire thickness of the nail plate (Fig 310) As the nails grow the transverse furrows slowly travel distally Like the lines of Reil they follow a great variety of acute febrile or systemic diseases and their distances from the cuticle edge permit estimation of the time which has elapsed since the event which caused their development (3–4 mm per month)

CHANGES OF TEXTURE

Either accompanying irregularities of the surface or occurring independently separation of parts of the nail substance from the plate may develop This may be called *desquamation* if the detached fragments are no larger than dust or bran (Fig 311) If the parts are larger and peel off horizontally the desquamation may be referred to as *lamellar exfoliation* (Fig 312) which already represents a minor degree of onychoschisis or horizontal splitting of the nail plate into two lamellae (Fig 313) Still more common than superficial detachment of scales is deep splitting of the nail substance in the *longitudinal direction* frequently starting at the free edge (Fig 314) In this case we speak of *splintering brittleness* or *onychorrhexis* (the technical terms of nail disorders are not very precise and are used by different authors with different meanings) Splintering of course may affect the whole surface of the nail and may be combined with irregularities longitudinal ridges and depressions (Fig 315) Splintering may also be very coarse and lead to deep longitudinal tears and splits (Fig 316) The thickened nail plate or parts of it may even break up into irregular pieces and granules, to present a *crumbly* appearance (Fig 317)



Fig. 298.—Longitudinal ridges (senile nails)



Fig. 299.—Longitudinal ridge and groove (due to a scar in the root of the nail)



Fig. 300.—Longitudinal grooves, due to fibroma in the nail wall in tuberous sclerosis



Fig. 301.—Longitudinal grooves with splintering (onychodystrophy)

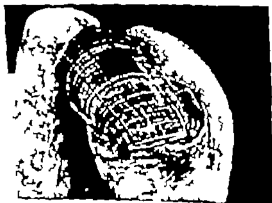


Fig. 302.—Longitudinal and transverse grooves (onychogryposis)

Shedding of scales lamellae splinters, and crumbs may cause a large part of the nail bed to become bare (Fig 318) and thus end in partial or even total absence of the nail (anonychia)

HYPERKERATOSIS

Thickening of the nail plate is not necessarily associated with changes in its surface and texture (e g the enlargement and thickening of the nail in acromeg



Fig. 303 —Pitted nails (psoriasis)



Fig 304 —Pits in longitudinal rows (psoriasis)



Fig 305 —Pitted nail with very few pits (psoriasis)

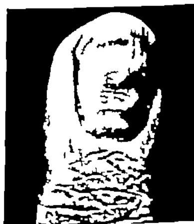


Fig 306.—Pits and trough-shaped irregularities of the surface (eczema venenatum from starch)

aly or the slight thickening in a case of pemphigus, Fig 319) To be sure discolorations, turbidities irregularities of the surface etc are in most cases at least to some degree associated with an increase in nail thickness (Fig 320) In some cases the most thickened extreme portion of the nail appears to be rolled at the edges making the distal part narrower than the proximal part (Fig 321)

If the hyperkeratosis of the nail forms lumpy masses or even long horn-shaped excrescences (Fig. 322) one calls the condition *onychogryposis* (onychogryphosis) (Greek *grypoo* to bend, or Latin *gryphus* a griffin) The surface of such a nail is frequently furrowed in both directions (Fig 302) A more or less *columnar* hyperkeratosis of the finger and toenails with thickened, wax colored, opaque and yellowish horny substance (Fig 323) is called *pachyonychia* (Greek *pachys* "thick")



Fig 307 —Central rough spoon nail (koilonychia in hypochromic anemia)

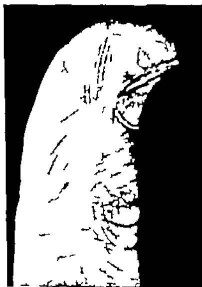


Fig 308 —Deformation of the fingertip in koilonychia dystrophica

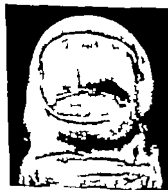


Fig 309 Transverse groove with scaly edge (Beau line in sclerodermia)

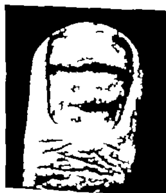


Fig. 310 —Transverse split (Beau line in erythroderma following iodine dermatitis)

Shedding of scales lamellae splinters and crumbs may cause a large part of the nail bed to become bare (Fig 318) and thus end in partial or even total *absence of the nail* (anonychia)

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Thickening of the nail plate is not necessarily associated with changes in its surface and texture (e g the enlargement and thickening of the nail in acromeg



Fig 303 —Pitted nails (psoriasis)



Fig 304 —Pits in longitudinal row (psoriasis)



Fig 305 —Pitted nail with very few pits (psoriasis)

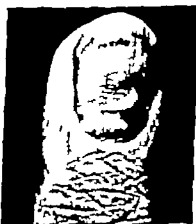


Fig 306.—Pits and trough-shaped irregularities of the surface (eczema venenatum from starch)

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Fig. 316.—Splintering with longitudinal splits (trichophytosis)



Fig. 317.—Crumbling (eczema)



Fig. 318.—Partial anonychia due to lamellated desquamation (trichophytosis)



Fig. 319.—Minor degree of hyperkeratosis (after bullae of pemphigus)



Fig. 320.—Hyperkeratosis (favus)



Fig 311.—Ordinary desquamation of the nail plate (eczema)



Fig 312.—Lamellated desquamation (koilonychia in iron deficiency)



Fig. 313 —Total lamellated desquamation, onychoschisis (koilonychia in iron deficiency)



Fig 314 — plintering in alopecia areata



Fig 315 —Splintering with longitudinal grooves and trough (onychodystrophia congenitalis)



FIG. 316.—Splintering with longitudinal splits (trichophytosis)



FIG. 317.—Crumbling (eczema)



FIG. 318.—Partial anonychia due to lamellated desquamation (trichophytosis)



FIG. 319.—Minor degree of hyperkeratosis (after bullae of pemphigus)



FIG. 320.—Hyperkeratosis (favus)



FIG. 311.—Ordinary desquamation of the nail plate (*eczema*)



FIG. 312.—Lamellated desquamation (*koilonychia in iron deficiency*)



FIG. 313.—Total lamellated desquamation onychoschisis (koilonychia in iron deficiency)



FIG. 314.—Splintering in alopecia areata



FIG. 315.—Splintering with longitudinal grooves and trough (onychodystrophia congenitalis)

telangiectases) The nail appears shortened because the cuticle usually grows over the nail much farther than is normal. Atrophic nails also generally exhibit exfoliation (onychoschisis) or splintering (onychorrhexis) from the free edge inward If there is nothing left of a nail plate except some insignificant lamellous or crumbly remnants, one speaks of *anonychia* (absence of nail Fig 327) In this condition the nail bed may be shrunken down to an irregular depression (Fig 328) or it may be blurred by atrophy or even be completely gone (Fig 329)

ONYCHOLYSIS

A frequently occurring phenomenon is *onycholysis detachment of the nail plate* from the nail bed Often it starts at the free edge and penetrates deeper in the center than at the sides, thus becoming crescent-shaped (onycholysis semilunaris, Fig 330) In other cases the nail becomes detached at one (Fig 331) or both *sides* (Fig. 332) The detached part of the nail appears partly opaque and light colored because of the reflection of light by air under the nail and partly dark because of dirt particles. The nail plate is usually of normal thickness, but a hyperkeratotic nail may also become loose similarly

DISTRIBUTION AND EXTENSION OF THE CHANGES WITHIN THE NAIL

Like skin and hair changes, pathological changes in the nails may be *disseminated* (pits in psoriasis) or diffuse (leukonychia totalis) They often have a pronounced tendency to occur in transverse (leukonychia striata) or longitudinal bands (senile ridges) They may affect only a part of the nail or the entire nail. *Partial changes* in a nail may start from *the free edge* (splintering and onycholysis) or they may originate at the opposite end at *the root of the nail* (desquamation, Fig 333 or defect Fig 334) and slowly shift distally Changes beginning at the proximal end of course, always indicate a disturbance of the matrix or of the nail wall, which in such cases, should receive special attention (paronychia, tumors, psoriasis, eczema, Fig 335)

CONSISTENCY OF THE NAIL

In examining nail diseases, *palpation* plays a minor role compared with inspection Nevertheless, there may of course, exist great differences in the consistencies of nail plates. In onychogryposis the nail substance is usually extremely *hard* *Extreme softness* and flexibility of the nail plate are encountered not only in thinning of the nails but also in marked hyperhidrosis (hapalonychia) *Loosening* of the consistency of the nail plate manifests itself by scales splinters, or crumbling so that actual palpation and methodical scratching are rarely necessary

DISEASES OF THE STRUCTURES SURROUNDING THE NAILS

A thorough examination of the nails, of course, also requires attention to the surroundings of the nails One searches especially for *skin diseases* which invade

Hyperkeratotic masses may also form *under* the nail plate They may grow from the hyponychium and appear as a granular or bulging layer at the free edge (Fig 324) This keratosis of the nail bed or of the hyponychium is termed *keratosis subungualis* It is mostly dark gray or blackish and occurs in a great variety of chronic inflammations of the nail bed and in diseases of the nails in general Keratosis subungualis may somewhat detach the free end of the nail plate and cause it to bend upward



Fig 321 —Hyperkeratosis with distal curling f edge

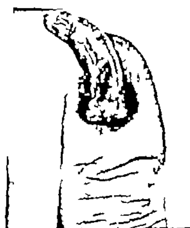


Fig. 322 —Onychogryposis, i.e., marked hyperkeratosis.



Fig. 323 —Pachyonychia



Fig 324 — ubungual keratosis (keratosis hyponychii)

ATROPHY AND ANONYCHIA

Much rarer than thickening is *thinning* of the nail plate (Fig 325) This condition can frequently be observed in atrophic states such as X ray atrophy of the terminal phalanx of a finger (Fig 326) where the surrounding skin also manifests clear signs of radiation injury (atrophic glossiness crinkled surface

telangiectases) The nail appears shortened because the cuticle usually grows over the nail much farther than is normal Atrophic nails also generally exhibit exfoliation (onychoschisis) or splintering (onychorrhexis) from the free edge inward. If there is nothing left of a nail plate except some insignificant lamellous or crumbly remnants, one speaks of *anonychia* (absence of nail Fig 327) In *this condition* the nail bed may be shrunken down to an irregular depression (Fig 328) or it may be blurred by atrophy or even be completely gone (Fig 329)

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A thorough examination of the nails, of course also requires attention to the surroundings of the nails One searches especially for *skin diseases* which invade



Fig. 325.—Atrophia ungulum (atypical Buerger's disease)



Fig. 326.—Atrophia ungulum (poikilodermatic X ray atrophy)



Fig. 327.—Anonychia with preserved nail bed (trichophytosis)



Fig. 328.—Anonychia with remnants of the nail bed (epidermolysis bullosa dystrophica)



Fig. 329.—Anonychia with vanishing nail bed (epidermolysis bullosa dystrophica)



Fig. 130.—Onycholysis of the free nail end (onycholysis distalis semilunaris).



Fig. 131.—Onycholysis of the lateral edge (psoriasis).



Fig. 132.—Onycholysis along both lateral edges (psoriasis).



Fig. 133.—Desquamation on the nail root (eczema).



Fig. 134.—Partial loss of nail plate near root (paronychia).



Fig. 135.—Vesiculo-squamous eczema on the root of the nail.



Fig 336.—Swelling f the nail wall (paronychia)



Fig 33 —Tumor of the nail wall (fibroma in tuberous sclerosis)



Fig 338 —Verrucae vulgares along the nail all and nder the nail

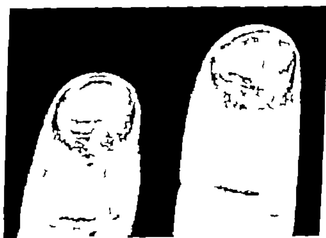


Fig. 339 —Elongated eponychium (ichthyosis vulgaris)

the nail itself from the nail wall (psoriasis, eczema, Fig 335) or form a common disorder together with the nail such as occurs for instance in X ray atrophy (Fig. 326) It is important to find out whether or not the *nail wall* is specifically altered, especially swollen as is the case in eczemas paronychias (Fig. 336) and tumors (Fig. 300) Tumors may be situated at or under the free edge of the nail (Fig 337) as is frequently the case with warts (Fig 338)

Besides the nail wall the *cuticle* (eponychium) also deserves attention because this structure may be altered in various nail and skin diseases (ichthyosis,

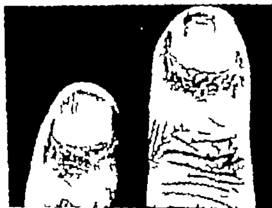


Fig 340 —Desquamating eponychium (scleroderma diffusum)

lupus erythematosus sclerodactylia) It may for example be widened (Fig 339) or cracked (Fig 340)

It need not be emphasized that *nail diseases may have connections with skin diseases* at distant sites (e g nail changes in alopecia areata) and that occasionally they may appear as *manifestations of internal diseases* (e.g koilonychia in hypochromic anemia due to iron deficiency) The physician who knows these connections is not likely to overlook them.

CHAPTER FIVE

Systemic Symptoms

LIKE other organs the diseased skin affects other parts of the body and is in turn affected by them Not only may a skin disease cause general phenomena and symptoms in other organs, but disease of other organs may also become the cause of skin disease. Such however is not invariably the case and while it is theoretically good advice always to consider cases in terms of "the entire person and to think of possible associations with changes beyond the skin for practical purposes the skin is *an organ* which like other organs, may often become diseased independently These independent skin diseases represent the essential part of dermatological practice and after all are the main reason why dermatology became separated from internal medicine to which it once belonged

Among the organs which are affected by skin disease the regional lymph nodes of the skin lesion in question must be considered first because of the frequency of their involvement. Such involvement is to be expected in the case of the infectious diseases Regional lymphadenopathies furthermore, play an important part not only in the diagnosis of some diseases (herpes syphilitic chancre Fig 341) but also occasionally in apparently non infectious dermatoses such as prurigo and even alopecia areata They may also sometimes require special therapeutic attention (ulcus molle skin tuberculosis carcinoma, pyoderma Fig 342) Sometimes dermatoses are associated with generalized lymphadenopathy (secondary syphilis leukemic eruptions)

Other organs are only rarely affected in the wake of skin diseases. We may find such complications in dermatoses which resemble septic states (nephritis and pneumonia in acute lupus erythematosus) and only exceptionally in more harmless infections of the skin (nephritis in impetigo infantum)

General symptoms are also not common in dermatoses except for exhaustion and nervousness following insomnia from itching Thus even generalized and universal dermatoses only occasionally cause *fever* (erythroderma pemphigus) Some skin diseases which start in a localized manner cause systemic phenomena only in far-advanced stages (furuncle carcinoma of the skin)

Only few skin diseases take a *fatal course* This is common in pemphigus mycosis fungoides very extensive burns certain rare neoplasms (sarcomatosis)

and blood dyscrasias with skin manifestations. Some skin diseases which are generally of benign nature may in exceptional cases end fatally like lupus erythematosus with acute exacerbation generalized erythrodermic psoriasis with severe arthropathy or carcinoma if it is treated too late and without success.

In view of the well-known mutual influences among all body organs, a skin disease may also be part of a more general disorder. This is true however only in a minority of cases, but when it occurs, it is of the greatest importance for diagnosis and treatment. In some cases the skin disease is simply a manifesta



Fig. 341 — Regional lymphadenopathy in syphilitic primary lesion



Fig. 342 — Regional lymphadenopathy in pyoderma of the head

tion of a *systemic infection* and either is its most striking feature (leprosy primary and secondary syphilis smallpox measles scarlet fever) or is only a less conspicuous or not regularly occurring sign (typhoid fever sepsis meningitis) In other cases the skin disease is the effect of a disease of the *blood-forming organs* (leukemic exanthems glossitis in pernicious anemia) or of *endocrine disorders* (myxedema cutis hyper and hypotrichosis intertriginous eczemas in obesity) Occasionally the skin disease may be due to an abnormal chemical composition of the blood (xanthoma in hypercholesteremia hydroa aestivalis in porphyria koilonychia in patients with hypochromic anemia and achylia gastrica associated with iron deficiency Fig 307 p 177)

Much has been said and written about the significance of the *psyche* in the pathogenesis of skin diseases especially those with itching and this subject has frequently been exaggerated in a fantastic manner The fact that *erythemas* may be provoked by embarrassed modesty *pallor* by fright and *sweating* by fear and even that established virus infections like warts can frequently be cured by suggestion does not prove that *nodules* and *blisters* can be psychogenically produced The well known skin changes observed in hysterics certainly have another genesis They are artifacts The few publications according to which lesions especially blisters have been produced by hypnosis are by no means sufficiently well documented or corroborated and even if they were their great rarity would render them insignificant. In most itching skin diseases, *the sequence of nervousness and disease is reversed* The patients do not get itching lesions because they are nervous (in which case the sanitaria should be full of itching patients) but rather it is much more likely that they become *nervous because the itching deprives them of rest and regular sleep* Especially various forms of atopic dermatitis (late exudative eczematoid Rost prurigo Besnier) have frequently and even very recently been claimed to be caused by *nervous influences* This is certainly not true. We have regularly observed the healing of these eczemas without additional treatment or hospitalization only to be followed by prompt recurrence a few days after discharge (Fig 346 p 242) We have seen this sequence of spontaneous cure and recurrence in many more than a hundred cases. The automatic certainty of this sequence of events in patients of all age groups including infants furnishes adequate proof in my opinion that psychic factors which are always changing and capricious have no decisive influence on it

There are also those skin changes whose development is connected with *localized disease of other organs* Good examples of this kind of association are acanthosis nigricans accompanying an internal carcinoma hypertrichosis due to ovarian and adrenal tumors disseminated telangiectasias and palmar erythema in cirrhosis of the liver erythema of the fingertips in chronic arthritis and symptomatic pruritus in dyspneic conditions (emphysema cardiac decompensation) In some cases the presence of another disease may change the course

of a dermatosis, as is the case of furunculosis in a diabetic, which frequently becomes especially severe. Other cases in point are the appearance of skin diseases (e.g. erythema exudativum multiforme, lupus erythematosus) following foci of infection in dental roots, tonsils appendixes, or elsewhere In these cases the causal connection frequently remains only hypothetical.

The simultaneous occurrence of a skin disease and a disorder of another organ is not always based on a cause-and-effect relationship. Both may be a *co-ordinated* effect of another cause or primary disturbance. This, of course is the case in systemic infections like syphilis and leprosy and also in Boeck's sarcoid if the disease affects the skin, lymph nodes, lungs eyes, and bones. We may also encounter such co-ordinated diseases in congenital anomalies. Thus congenital absence of sweat glands may be associated with hypotrichosis rhinitis atrophicans with saddle nose and brachycephalism with dental defects and occasionally even imbecility In von Recklinghausen's disease, tumors of the peripheral nerves psychic disorders (mental retardation, reduced libido) and malformation of bones may occur together with the skin changes (pigmented spots, tumors) In tuberous sclerosis, tumors of the facial skin (adenoma sebaceum Pringle) nail walls, and gums coexist with tumors of the kidneys heart, and gastrointestinal tract in addition to mental retardation and epileptiform seizures It has been proposed that these two diseases be classified as diseases of the ectodermal system" because not only the skin but all derivatives of the ectoderm seem to be affected. This conception however is still too narrow because even such entirely mesenchymal structures as bones have also been found to be affected. Skin diseases which are due neither to infection nor to developmental disturbance may also be associated with changes in other organs though nothing tangible is known about the modes of connection. For example this holds true in psoriasis (unless one considers it to be an infectious disease) because a small fraction of the cases is associated with a definite clinical variety of chronic polyarthritis. It also applies to certain papulovesicular and lichenified eczemas (atopic dermatitis prurigo Besnier neurodermatitis) which are not infrequently associated with asthma. However a direct relationship of both chronically recurrent diseases in the form of simultaneous exacerbation or regular alternation cannot be established.

It is of great importance to get a true conception of the *frequency* and *practical significance of the relationship* between dermatoses and other diseases. In the old Hippocratic and Galenic teaching of medicine skin diseases were considered entirely as phenomena accompanying the elimination of bad humors from the body system a conception of which the term *eruption* is still reminiscent Skin diseases were in a sense considered as phenomena of healing with the skin functioning as an organ of excretion like the kidney It was two thousand years until Hebra freed us from this humoral pathologic nonsense which had completely paralyzed dermatologic and therapeutic research In 1844 by his

thorough study and analysis he dispelled *the old belief in the existence of a* scabietic constitution or dyscrasia a belief which was stubbornly adhered to in *spite of the fact that* the deliberate production of scabies by transfer of the parasite had already been attained as early as 1786 It was after Hebra accomplished this that the ***skin was recognized as an organ*** like the kidney or the stomach and that it could have its own diseases and not be merely the mirror of disease in other organs. This revolutionary concept led to the differentiation of ***idiopathic*** and ***symptomatic*** dermatoses the former being localized in the skin organ and the latter being produced by internal processes with the skin as an effector of some disturbance elsewhere. It would seem of greater practical importance to divide the ***independent*** skin diseases those with pathological changes restricted only to the skin from what might be called ***correlated*** dermatoses. It would then be *of secondary importance in the latter to decide whether* the skin disease is the effect of the internal disease or vice versa whether the internal disease results from the skin disease or even finally whether the relationship between the two remains unknown In any case whenever ***statistical correlations of frequency*** between a dermatosis and other disorders have been demonstrated one must give thought to this fact in diagnosis as well as in therapy

In order to indicate the relationship of a disease to certain other characteristics the term ***constitution*** has often been used and the attempt has been made to construct a 'pathology of constitution which however should always be based on statistically demonstrable relations. Constitution is the sum total of these relations. If the statistical proof of these relationships is lacking it is without practical value to talk about constitutional properties which predispose to or protect against, certain diseases.

The fact that some dermatoses have connections with other diseases should not cause physicians to impose on patients unnecessary examinations or bother them with useless diets and other general methods of treatment A good dermatologist should know ***which dermatoses*** have connections with other diseases so that he will be careful not to overlook anything of that sort On the other hand he should be fully aware that in the majority of the cases a connection between skin diseases and other disorders is ***sought in vain time and again*** *It may* of course be true that a certain form of eczema in a considerable number of cases is associated with asthma psoriasis with arthritis and vitiligo with nervous disease Certainly *we also occasionally* see *lupus erythematosus* become acute and systemic and it may possibly even be true that patients with acne more frequently suffer from chronic constipation than do others *In spite* of all this the main fact in the foreground remains that the overwhelming majority of all sufferers from eczema psoriasis vitiligo lupus erythematosus acne etc are for all practical purposes otherwise ***completely healthy individuals*** In these conditions it thus is usually justifiable to speak of diseases of

healthy men But even if statistically sound correlations with internal disorders can occasionally be established their entire practical value still depends on their *order of frequency* We should guard ourselves against accepting the fantastic exaggerations which today as in Hebra's times are the vogue of constitutional pathology These claims pose as modern but in reality they are old fashioned and reactionary Of course we should not cease to search in all skin diseases, especially in the less-well-known ones for associations with diseases of other organs. But in doing so our motto should be *Always think of it but be reluctant to believe it.* Thus there is no proof that psoriasis is associated with a disorder of sugar metabolism that ichthyosis and nevi are based on parental syphilis that the telangiectases along the costal arch indicate heart and lung diseases that ruby spots (angiomata senilis) point to internal cancer or that red-haired individuals are liable to show deficiencies of the sensory organs and even of the psyche. Even in the case of hypertrichosis, which of course may be a very typical symptom of endocrine disorders, one only *rarely* succeeds in establishing a connection with such endocrine disturbance. Xanthomatosis may be caused by hypercholesterolemia and yet there are other clinical types of xanthomatosis with completely normal blood cholesterol levels. Hydroa aestivalis is a sensitivity of the skin to light associated with porphyria and yet there exists a different type of sensitivity to light, xeroderma pigmentosum *without* this metabolic anomaly The "pathologists of constitution of course believe that they may miss something if they do not keep on searching for abnormal porphyrins in xeroderma patients. It is also remarkable how rarely disorders of metabolism or the nervous system even of the most serious types cause skin diseases. On the other hand the skin in spite of its roles in water elimination and temperature regulation, is practically without significance in the pathology of metabolism. Even the general condition of the *skin itself* its pigmentation vascularization, dryness, hair growth, etc. is in most cases not related to the occurrence of specific skin diseases. There are, of course some examples of correlations between certain types of skin and certain skin diseases (erythema solare and ruddy skin arsenical melanosis and brunet complexion) or between two different skin diseases (ichthyosis and atopic dermatitis)

The decisive question to be asked of constitutional pathology is whether or not the relationship of a skin disease to other organs provides a guidepost for *therapy* This point belongs to the chapter on general therapy of skin diseases where it will be discussed.

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deep trichophytosis and tertiary syphilis remain relatively indolent. The pain sensation elicited by pressure with a small probe in small to nummular lesions is in some diseases (secondary syphilis lupus erythematosus) particularly vivid and sharp

Very striking is the hyperalgesia on the slightest touch in the eruptive stages of herpes zoster Later it may be replaced by analgesia. Because the skin manifestations in this condition depend on an infectious inflammation of a spinal ganglion pronounced *neuralgic pains* may also occur Especially in old people these pains may continue even *after* healing of the skin eruption causing unbearable agony

Analgesia (absence of pain sensation) which may come about as a sequel to diseases of the central nervous system is only rarely important in dermatology (ulcus perforans)

Hyperesthesia and hypesthesia or *anesthesia* refer not only to disturbances of pain sensation but also to those of *temperature sense* (syringomyelia) and *sense of touch* (sclerodactylia keratoses) Localized anesthesia and hyperesthesia are of great importance in the diagnosis of leprosy especially the tuberculoid form.

The *sensation of heat* is an important accompaniment of many localized inflammatory processes. If excessive, it may become burning and painful. Some times in purely angioneurotic dilatation of the cutaneous blood vessels the sensation of heat may cause pronounced discomfort, especially in emotional erythema of the face or in rosacea.

On the other hand some skin patients suffer from a *sensation of cold* Even the normal person has a disagreeable sensation from cold and responds with constriction of cutaneous blood vessels (anemia) cyanosis cutis marmorata, and contraction of the arrectores pilorum muscles (cutis anserina, goose flesh) In some diseases a localized sensation of cold exists (acrocyanosis, sclerodactylia Raynaud s disease) In extensive inflammatory states of the skin (generalized eczema erythroderma) a marked general tendency to feel cold and to shiver is noted Therefore in examining such patients one should avoid unnecessary exposure. However most dominating among the subjective complaints of skin patients are the paresthesias particularly the special type of paresthesia known as *itching* It has been said that itching is a usually unpleasant sensation which elicits the desire to scratch. This definition however is not quite correct for there are types of itching which do not evoke scratching as does the *scratch itching* of papular eczemas strophulus, and scabies but which rather are relieved by pressing rubbing, or pinching The latter kind of itching occurs in some cases of urticaria, keloids and lichenified eczema. The term *rub itching* may be used to characterize this phenomenon which does not lead to scratch marks but rather to suggillations or bruises (Fig 343) Itching usually occurs periodically and frequently in distinct severe attacks, especially on disrobing in the evening and also more frequently and violently during the night

CHAPTER SIX

Subjective Symptoms and History

IN CONTRAST to the situation in other disciplines of medicine subjective symptoms are rarely of diagnostic value in dermatology Of course such symptoms are extremely important from the therapeutic viewpoint because the patient s foremost desire is to obtain relief from suffering The sympathetic physician therefore will concentrate his therapy especially on the subjective complaints Frequently the patient is prompted to visit his physician by either the *fear* that an asymptomatic, but plainly visible change may turn into a serious matter or by the *handicap* caused by the cosmetic disfigurement. In considering the latter it would be a mistake to believe that things which are neither painful nor dangerous are not of much importance By their unusual and even repulsive appearance alone skin diseases may have the most disastrous consequences for the patient They may torture him with anxiety drive him out of his job destroy his marriage—indeed may make him an outcast in any human society Therefore the physician should not judge cosmetics lightly thinking only of makeup nail polish or dyed hair A case of psoriasis without any subjective complaints or a case of mycosis of the nails may destroy a young girl s chances to get married thus fatefully stunting a hopeful life. A harmless birthmark on the face or a total alopecia may make the afflicted person completely unfit for many vocations A healed and asymptomatic destruction of the nose or face due to lupus vulgaris or syphilis can render the marked person frightful to children and repulsive to grownups Even without subjective symptoms the skin patient frequently is a most pitiable individual urgently in need of all possible medical care

Subjective complaints may be of different kinds In the first place skin diseases like all other diseases may be painful *Hyperalgesia* may exceptionally accompany non inflammatory skin lesions (leiomyoma) but is most common in inflammations It may therefore be of value in the differentiation of inflammatory from non inflammatory disorders (abscess versus cyst) Different inflammatory skin diseases may possess very different degrees of painfulness on pressure thus furnishing clues for a differential diagnosis The tuberculous ulcer and the soft chancre are sensitive to touch the syphilitic chancre is not. The swellings of furunculosis are usually very painful while the infiltrates of

in forbidding the patient to scratch. The patient is especially helpless against scratching while asleep. Therefore the physician should not add to the patient's troubles by giving orders which he cannot obey. He had better do what he can to stop the itching. Itching can be so tormenting that the patient has a feeling of relief from the pain which supersedes itching after furious scratching. Itching can drive the tormented sufferer to suicide. The attitude of the layman who has a healthy respect for pain but is inclined to look upon itching as something funny should never be shared by the physician.

If itching is centrally caused or if its cause is located in the nervous pathways, it will of course not be relieved by scratching. Also the itching of the mucosae usually cannot be influenced by scratching and therefore may be extremely



Fig. 345 Holes in the shirt from scratching in intense pruritus (poikiloderma [illegible])

tormenting. Objectively itching can be diagnosed from the scratch marks and in some cases also by the effects of rubbing or pinching. In extensive chronic, itching eruptions the nails appear rubbed short and have a glossy polish (glossy nails, Fig. 297, p. 173). Occasionally the underwear is scratched through (Fig. 345). If there are no scratch marks present, the physician may recognize or guess pruritus from the presence of a skin disease which is pruritic without leading to scratch marks (urticaria, lichen planus). Conversely he will know immediately that itching is not a symptom in many other diseases. Whether or not pruritus is present, therefore, is usually deduced from the appearance of the skin rather than by questioning the patient. In diagnosis by the dermatologist, the history often need not play so important a role as it does in other medical disciplines. Since the object of this diagnosis is placed immediately

than during the day Once the attack has started in one place it usually spreads into its surroundings. It may even jump into the healthy skin of distant areas so that for example a patient with an itching eczema of the leg may eventually also scratch and excoriate the healthy skin on the back (Fig 344) Itching thus is a radiating sensation Of course psychic factors may influence this phenomenon of so-called radiation Psychic factors alone are able to elicit itching (itching from merely looking at a patient with pediculosis) The urge to scratch may be irresistible even imperative so that there is little point



Fig. 343 — [illegible]ations resulting from pinching in pruritus

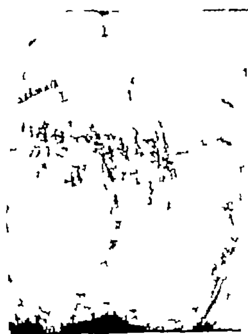


Fig. 344 —Distant itching Excoriations with otherwise unchanged skin of the back in patient with itching eczema of the lower leg

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in front of his eyes the dermatologist may even be advised to forego any questioning in the beginning and to permit the *pathological picture to tell the story* The skin divulges what it has to say with eruptions and not with words. If the physician has once listened attentively to the language of the skin there generally remain only *two questions* of value for the diagnosis namely How long? and Does it itch? Even these two questions are mostly asked only to *confirm what the examining physician already knows* The experienced skin specialist can readily estimate *how long a time* a disease has been in existence He will not only appraise the character of the lesions the bright red color of acute hyperemia *and the livid hues of chronic hyperemia but also consider the* typical accompaniments of longer-established processes such as pigmentary changes and scars He also knows immediately whether or not a disease *itches* If it itches it will have been scratched and show evidences of it (erythematous streaks excoriations glossy nails) or it is likely to have been rubbed (glossiness rubbed-off hair) or pinched (suggillations) The *familial* history which plays such an important part in other medical fields (e g in internal medicine) as a rule contributes little to the diagnosis especially in the case of eczemas because *we know so little tangible about their heredity Here again scabies is in an* exceptional position Scabies is probably the only skin disease in which in doubtful cases the family history and still more important examination of the family may be diagnostic It goes without saying that in contagious (favus tuberculosis) and hereditary diseases, family and contacts at home should not be forgotten *But these are exceptional instances In general for the dermatologist* conversation about history has less diagnostic than psychological importance It allows him to show his interest in the patient and to establish contact with him

Exceptions are all cases in which special *exogenous* factors may be of importance (traumatic medicinal cosmetic occupational climatic or nutritional) so that their clarification is a prerequisite to an adequate diagnosis. In these conditions the history must be taken very conscientiously and comprehensively

Entirely different is the value of dermatologic history taking with regard to *therapy* In the case of patients with chronic skin diseases who have already been treated with many ointments tablets and injections, nothing in the beginning is more important than a most detailed *therapeutic history* which on the one hand uncovers the gaps in the past treatment and on the other hand permits one to find a foundation of *well tolerated ointments, bases, and medications* on which future treatment can be built It is particularly important that the physician procure exact information about previous X ray treatments since the *decision* as to whether further radiations are desirable or possibly contraindicated depends entirely on the dose and quality of the former radiations.

CHAPTER SEVEN

Auxiliary Diagnostic Techniques

During the clinical examination we make use of various technical aids to sharpen our observations. The *diascope* is a particularly indispensable instrument for the dermatologist, who should always have one handy. Considering the small size of many lesions, a magnifying glass is almost as important. *Binocular loupes* or spectacle loupes such as ophthalmologists use are very practical especially in cosmetic minor surgery. Higher magnifications which so far have remained without much practical importance can be obtained with the *dermatoscope* a device with a built in source of illumination.

Needle and *scalpel* are sometimes required to differentiate vesicles from solid lesions by puncture. A fine blunt *probe* is practical in testing the consistency of small lesions as well as their tenderness or hyperalgesia. In France, special little dull *knives* are recommended with which to scratch scales off methodically (*grattage méthodique*). Though this method is cleaner than scratching with the fingernail the latter is preferable because it provides better sensory findings.

Special *lighting* of the lesions being examined may in exceptional cases lead to results of diagnostic importance. *Wood light* produced by a special filter attachment to a source of ultraviolet light is frequently used because it makes some pathogenic fungus infections in the hair visible by fluorescence. The Wood light is also important in such cases during therapy to evaluate the results of the treatment.

Besides the afore-mentioned clinical aids, there are of course also *laboratory methods* which are useful for the diagnosis of skin diseases. It has been estimated that in a dermatologic clinic scarcely 2 per cent of the cases need such laboratory examinations in order to make a diagnosis. The *clinical* examination thus retains *prime* importance particularly since it is well known that laboratory tests may lead one astray if they are not evaluated in the context of the clinical picture. On the other hand, the laboratory frequently has the last word when clinical methods alone fail to arrive at a diagnosis. Often too they are indispensable for ascertaining or confirming the diagnosis which the clinician has already made. Therefore, the skin specialist like any other physician, must be on guard, if the diagnosis is not completely secured not to omit laboratory

examinations simply because they are complicated and expensive The dermatologic clinician should be familiar with the various specific laboratory methods available and their effectiveness

The clinical examination alone with its exact observation palpation scratching and puncturing of the lesions amounts essentially to "pathologic gross anatomy with the naked eye (Darier) This is extended into pathologic histology by the *biopsy* a method which can be so easily used in no other specialty as in dermatology The easy accessibility of the object for biopsy permits the use of a *variety of methods* *The classical method is oval excision* followed by suturing The cut is made across the border line between healthy and diseased skin so that the removed specimen contains both normal and pathological tissues for comparison In superficial skin diseases it is simpler to lift a small skin fold with forceps freeze it with ethyl chloride, and then snip it off with *curved scissors* However the most important specifically dermatologic method is excision with the Kromayer *punch* a small tubular knife which by rotating movements between thumb and index finger cuts through the entire thickness of the abnormal skin The resulting cylindrical piece of skin is lifted out with forceps and cut off at its base with pointed scissors. This small operation is done under local anesthesia If the minor bleeding has been stopped by *touching with liquor ferri no other care of the wound is necessary* except that some skin-colored antiseptic powder may be applied Adhesive tape is usually unnecessary The punch method besides its simplicity has the great advantage that the patient does not have the impression of surgery and therefore is less likely to object to the excision One may truthfully tell him that a small bit of skin will be removed with a special instrument Therefore the Kromayer punch biopsy in general is the method of choice.

There are of course many cases in which the histopathologic examination is not of much value because it only confirms what clinical observation has already led us to assume about the pathological basis of the eruption in question In other instances however it may decide a difficult differentiation or even surprise us The especially valuable domain of the biopsy is the large field of tumors. Many neoplasms cannot be diagnosed at all without microscopic examination

Following biopsy in order of importance for dermatologic diagnosis are the *microbiological methods of examination and among them* especially the search for pathogenic fungi The simplest procedure and therefore the most commonly used is to clear the scales or hairs which have been removed for examination in 10 per cent potassium hydroxide and examine them with a dry moderately high-power microscope With this method the beginner may easily mistake the borders of cells for mycelia and he may also miss fungi which do not become visible until after several hours of soaking in potassium hydroxide. Therefore

It is sometimes also advisable to *stain* the specimens for fungi In the case of erythrasma this is always necessary because of the small size of the fungus elements.

Examinations for *cocci* and other *bacteria* are especially necessary in venereal diseases, leprosy and chronic ulcerations. They are done according to the well-known rules of general bacteriology

Frequently especially in dermatomycoses, *cultural investigations* are useful In recent years they have gained in importance in determining the sensitivity of bacteria to antibiotics, especially penicillin.

Diagnostic *animal inoculation* must also be considered for example in tuberculosis, glanders (guinea pig) sporotrichosis (rat) and anthrax (mouse)

Serological tests are used practically only in syphilis but *immune biological* methods play a part not only in the diagnosis of old syphilitic infections (luetin) but also in skin tuberculosis (cutaneous tuberculin tests) and in dermatomycoses (trichophytin tests) To find the exact causes of some urticarias and eczemas intracutaneous allergy tests are often made but their value frequently remains doubtful. More important is Jadassohn s *patch test* useful in the effort to uncover special sensitizations to occupational medicinal and other contact substances The test is done by attaching to the skin with adhesive *tape* a 0.5-cm in diameter linen patch with the suspected substance After 24 hours the patch is removed, and another 24 hours later the skin is checked for a reaction. The method often permits us to diagnose the specific etiology of a contact dermatitis, e.g to distinguish primrose dermatitis from dahlia dermatitis or mercury irritation from tar irritation. However the patch test may also lead us astray especially in occupational eczemas. If the reaction is distinctly positive, much time is gained In doubtful cases, however it is safer and more sound to test the patient by actual occupational exposure and if necessary to do this repeatedly

I bullous dermatoses the patch test is also used to diagnose a sensitivity to iodide which is common in dermatitis herpetiformis Duhring but rare in malignant pemphigus vulgaris.

Finally the necessity *for examining urine blood* or *spinal fluid* in certain dermatoses must be mentioned The testing of *urine* for porphyrins is especially necessary in the diagnosis of hydroa aestivalis the *blood count* and cell pathology in the diagnosis of skin manifestations of leukemia polycythemia, anemia (koilonychia) systemic lupus erythematosus, and pruritus and the examination of *spinal f id* in syphilis.

In exceptional cases it may become necessary to examine the *chemistry of the skin* (sugar cholesterol calcium) which can be done on punched-out specimens with histochemical or microchemical methods. Occasionally there is need for an eye examination (Von Recklinghausen s disease Boeck s sarcoid) The *nose*

should be routinely investigated in facial lupus vulgaris and hemorrhagic telangiectasia (Osler's disease)

X *ray examination* of bones may become necessary in lupus vulgaris, Boeck's sarcoid pseudo-xanthoma elasticum and Von Recklinghausen's disease It is also always the responsibility of the physician to think of all other examinations which may clarify the diagnosis and to select carefully the best ones to make sure that no obligatory tests are omitted On the other hand the patient must not be bothered unnecessarily or subjected to unwarranted expense.

GENERAL PRINCIPLES OF THERAPY

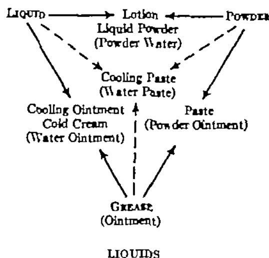
Introduction

WHILE dermatology frequently utilizes the same therapeutic methods as those used in internal medicine and in surgery it has developed *one* method as a specialty namely the *external application of drugs to the skin* This essentially dermatological method forms an integral part of the general therapy of skin diseases and therefore requires a full discussion. There are some dermatologists who think it is beneath their dignity to concern themselves adequately with external therapy Sometimes they even say that the whole business of treatment with salves is only a matter of not having anything better This certainly is a peculiar way to look at the matter If one is able to cure a skin disease as easily by internal treatment as by way of salves, no sensible physician will go so far as to stretch out his hand for the ointment jar Certainly swallowing tablets or receiving injections is a much simpler more convenient, and cleaner method for the patient than the use of greasy ointments, which requires much patience and effort from the patient. However as long as a number of the most common and important skin diseases respond more readily to external treatment or even can reliably be cured *solely* by external treatment the dermatologist must consider himself fortunate to be in possession of this method of therapy He must devote much attention to its study and he must take great trouble to counteract the frequent underrating and aversion to salves by the patient

CHAPTER EIGHT

The Vehicles

IT IS easy to obtain an understanding of vehicles by reducing them to their three basic elements namely liquid powder and grease (oil fat) Most vehicles consist of these basic ingredients or of mixtures of them. The following diagram shows the names which have been given to these mixtures



LIQUIDS

Water occupies the most important place among liquids used therapeutically It can be used for washings baths or compresses. Though washing or dabbing with water is occasionally used (intertriginous eczemas) washing in general plays only a *negative* role in the treatment of skin diseases. It happens time and again that the skin specialist has to caution the patient against unnecessary washing and cleansing of the diseased skin because it aggravates so many skin diseases especially the eczemas. Frequent washing-off of applied medicaments weakens their effects and the patient s manipulations on his eruptions in the process may make medical observation difficult or even misleading The risk connected with washing of diseased skin is increased by the use of soap Soaps are compounds of fats and alkalies Alkalies make the horny layer swell and thus damage the epidermis with their keratolytic and even caustic effect It is not sufficient to use *superfatted* soap to avoid such soap injury to the skin because even such soaps may still contain much alkali For these reasons washing is rightfully frowned upon in dermatology

By the same token, in the *prophylaxis* of skin diseases cleanliness achieved by washing and bathing and so-called personal hygiene do not play the important roles that the layman expects. More likely the opposite is true. Not even intertriginous eczema of the axillae and groins, which is frequently thought to be caused by irritation from accumulated secretions, can be prevented by washing. On the contrary it is my impression that cleanliness here is an unfavorable factor Powders and pastes which *do not remove* the feared secretions have a better effect I have never heard that vagrants and other people who do not believe in washing are specially subject to intertriginous eczemas.

With the exception of infections associated with gross dirtiness (pediculosis scabies pyodermas) skin diseases are by no means prevented but rather in many cases, are caused or aggravated by cleanliness and so-called hygienic procedures. We owe to the more sophisticated cosmetics many instances of dermatitis (eczemas from hair dyes, lipsticks day and night creams paronychias from manicures ugly pigmentations from cologne) Also bathing and swimming (mycoses of the feet, plant dermatitis) shaving (trichophytosis of the beard) and oral hygiene (mouthwash eczema) may create skin diseases. Excessive exposure to sunlight is also predominantly harmful since it sometimes causes skin diseases (solar dermatitis with melanoderma and leukoderma precocious wrinkling keratosis senilis with carcinoma) and sometimes leads to aggravation of existing disease processes (eczemas, lupus erythematosus psoriasis) Therefore there is good reason to say that the whole complex of personal hygiene—cleanliness, light, air sun bathing and beauty care—is more likely to be harmful to the skin than beneficial. It is unlikely however that, on account of this fact, people will decide to give it up

Nevertheless, it is easy to understand why in dermatologic therapy washings and *baths* are only exceptionally used. Because of their effect on the circulation, alternating hot and cold baths have been recommended for chilblains. *Lukewarm baths* sometimes have a place in managing pruritus because of their soothing effect on the nerves. The relief they produce however is very transient. The use of medicated baths which was once considered very important in dermatology has been practically abandoned. Various chemical additions to baths can be applied only in such weak dilutions and for such relatively short times that for example it does not seem logical to prescribe a sulfur bath when one can apply a 33 per cent sulfur ointment. Sulfur baths, tar baths, etc. can be prescribed only in the hope that certain medicaments may have a different effect on the warmed-up and wet skin than on the dry one. Nothing of this kind however has been empirically established so far in fact no one has even tried to examine the problem properly There has been some dermatologic interest

1 The soothing effect of warm "colloidal baths" with cornstarch or oatmeal cannot be denied. A cup of cornstarch is mixed with cold water and then blended with the warm bath of adult size. An oatmeal preparation called Aveeno is easier to mix. These slightly slippery baths (careful with

in baths in the form of Hebra's water bed. This is a permanent bath which is used when large parts of the body surface have been deprived of their protective skin covering (burns, pemphigus, decubitus, gangrene). Such a water bed may render bearable the life of the patient so afflicted by relieving pain, soothing, cleansing and deodorizing. However water beds have the disadvantages of requiring quite complicated installations, of favoring monilial infections in the case of some chronic dermatoses, or of making the patient so dependent on his water bed that he never wants to come out of it. Therefore water beds have not found general acceptance. The advent of corticosteroid therapy has further reduced the need for the water bed.

Of great practical importance in dermatology is the use of water for *compresses*. Strictly speaking, compresses do not represent so much a use of water itself as a method by which to produce warm or cool temperatures on the skin. Whether one accomplishes heating or cooling effects depends on the degree of free exposure to air of the dressing. *Impermeable or closed moist dressings* (covering with a plastic sheet, oiled silk or other impermeable films) heat up rapidly even if they are applied cold. They remain moist and therefore may stay in place for a long time, even overnight. In order to increase their heating effect a special source of heat may be placed over the impermeable covering (electric pad, hot water bottle). Closed moist dressings are used whenever a *depth* effect is desired as, for instance, in subcutaneous inflammations (boils, abscesses, deep trichophytosis). In *superficial inflammations* (acute eczemas), *open wet dressings* are recommended. It is a peculiar fact that, in this respect, the skin responds quite differently from other tissues. While it is common practice to treat inflammations with heat in order to stimulate defense mechanisms at the focus of inflammations by active hyperemia, such measures are wrong in epidermal inflammations. Eczemas do not tolerate heat. Therefore acute weeping eczemas are the domain of the air-exposed or open wet dressing. This procedure is carried out simply by placing moist compresses on the oozing skin and changing them before they get warm or dry out or stick to the skin (about every 5–10 minutes). Under the influence of gentle cooling from evaporation, oozing usually stops soon. The permeable or open moist dressings are indicated in all forms of acute oozing dermatitis especially, because they practically never irritate and therefore do not entail any risk. As soon as the skin has become dry, one starts to treat with lotions and pastes. If the dermatitis is no longer weeping, open moist dressings are generally useless. This means that cooling has little influence on dry subacute inflammations of the skin.

Of course, it is customary to add chemically active agents to the moist dressings. In closed (impermeable) dressings, liquor aluminii acetici (Burow's

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aged patients!) and also baths with a weak, wine-colored solution of potassium permanganate are very popular in America (Transl.)

solution) and in open dressings, 2 per cent boric acid or 10 per cent alcohol are commonly used. Camomile tea, a weak decoction of the flowers of *Matricaria chamomilla* is widely used for moist dressings in Europe but is little known in America, where the drug is seldom available in good quality The reason why some agents are preferred for open and others for closed dressings is unknown Alcohol (pure grain alcohol not denatured or rubbing alcohol) evaporates easily thus having a greater cooling effect than water It also does not leave an irritating concentrate. Boric acid is a weak disinfectant and astringent which hardly irritates, even in saturated solutions. Since the chemical action of the ingredients is negligible, it would seem quite natural to make open wet dressings simply with water If one wants to be sure of a completely bland treatment one may use isotonic (0 5–0 9 per cent) sodium chloride solution (normal saline solution) Whether hypotonic solutions (distilled water) or hypertonic solutions (e.g 2 per cent sodium chloride) differ in effect from isotonic sodium chloride is not known So far in my own investigations I have not been able to find a difference. If in stubborn cases, one wants to have not only a cooling but also an astringent effect, one may add to the solution ½–1 per cent solutio aluminii acetici 0 1–0.01 per cent potassium permanganate or 2 per cent tannic acid or in addition to the moist dressings, one may touch the oozing areas with 2 per cent silver nitrate solution. In this manner one may enhance epithelization in some cases. Of course it is prudent to start with completely bland solutions and to progress to chemically active agents if healing does not progress rapidly enough.

Open wet dressings are indicated in all acute inflammatory processes with loss of epidermal covering and marked exudation as long as there is no pyogenic infection with crust formation which would necessitate antibiotic ointment dressings In general, it is profitable not to apply open wet dressings uninterruptedly if for no other reason than not to interfere with the patient s occupation One may use them three times daily for 2 hours and cover the skin in the interval with a lotion or a zinc oxide oil

Rx Zinc oxide
Ol oliv aa 50 0

Cooling salves also usually work out well but may sometimes carry the risk of ointment dermatitis.

Besides water *dilute alcohol* is the other liquid most commonly used in the treatment of skin diseases, mainly for dabbing or wiping the skin. It removes secretions and irritants more easily and more gently than water It has a drying cooling and vasoconstrictive effect and therefore counteracts inflammations and relieves itching. If dilute alcohol makes the skin too dry one may add 5 per cent glycerin to it.

As a cleanser after the use of ointments, *benzene* with or without 30 per cent

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In some cases, chemically active agents are mixed with the powders

Drying and astringent terra silicea purificata, subgallate of bismuth and also tannic acid (e.g., equal parts of tannic acid and zinc oxide used in balanitis)
Caustic resorcinol (used in condylomata acuminata)
Antiseptic sulfur precipitatum, iodoform
Antipruritic menthol
Antihydrotic (*idros* Greek, "sweat") tannic acid, formalin

In order to make the use of powders more acceptable, pigments to blend with skin color (red cinnabar eosin, *bolus rubra* or *bentonite* with iron oxide and brown Bismarck brown ichthyol) may be added e.g., for the daytime treatment of acne. Such powders, of course are offered by the cosmetic industry but they are of little importance in dermatologic therapy

Indications for treatment with powders are burning and itching mechanical irritations (intertriginous eczemas, jock-strap itch chafing) and superficial inflammations with little exudation. They are also sometimes used as a covering for messy ointments or tars to make them stay on the skin

Occasionally powders may be used for cleansing. In such cases, starch may be applied on cotton to help soak up secretions or excess of oil which may have been used.

An important *contraindication* to the use of powders is the presence of any marked exudation, especially pus, because exudate and pus cement the powder dust into hard coarse granules and masses which irritate mechanically and *promote infectious processes, especially in hairy areas. Treatment with* powders is useless in all deeper and chronic processes unless the powders contain very strong chemotherapeutic agents.

The *application* of powders is done with gauze sponges cotton, or cotton sticks (swabs) Since only very small amounts of powder adhere to very dry skin, it is practical in such cases to dab the skin first with 5 per cent glycerin in 20 per cent grain alcohol which leaves the skin slightly sticky If powder is to be applied in large amounts, a powder shaker may be used. For the very copious use of powder the graphic terms powder shirt" and powder bed" are sometimes used.

SHAKE LOTIONS—LIQUID POWDER, SHAKE MIXTURE, DRYING LOTION SHAKE OINTMENT

One of the great shortcomings of treatment with powders lies in the fact that so little powder sticks to the skin. This *gave rise to the idea* of using a suspension of the powder in a mixture of glycerin and water as a paintlike covering In these suspensions the powder rapidly settles to the bottom so that they have to be shaken before use. They *are therefore* called *shake lotions* After application, they dry rapidly —whence the name *drying lotion*.

oil is preferable because it removes residual ointment with less rubbing and therefore is less likely to cause mechanical irritation

Dilute alcohol dabbings are indicated in the *first stages of eczema, provided* that there is no oozing because alcohol stings eroded skin. In some people the genitalia and eyelids also burn on contact with alcohol

The main shortcomings of alcohol dabbing are its superficial and transient effects. Therefore, alcohol is often not used alone but rather as a *solvent for medicaments* especially antieczematics (resorcinol tar) antipruritics (menthol phenol) and stimulants (bichloride of mercury iodine) Especially in hairy areas where ointments and powders are out of place chemical agents if possible are used in the form of alcoholic solutions (hair tonics tinctures) even though *it is a decided disadvantage that the hair tends to become dry and* brittle with their use. One may in these cases add 0 5–2 per cent of castor oil. Glycerin is not advisable because it makes the hair sticky Of course, besides alcohol other *liquids* such as ether chloroform acetone and benzol occasionally are also used as solvents chiefly for tars and chrysarobin

POWDERS

Dusting powders form a very loose and thin layer on the skin Since the depth effects of medicaments depend largely on their surface-occlusive qualities, *powders have only a very superficial effect If exudation is minimal* they are drying because their granules absorb moisture and permit quicker evaporation by exposing a larger surface. This like the open wet dressing has cooling vasoconstrictive and therefore anti inflammatory and with some exceptions, antipruritic effects. Powders also decrease friction between adjoining skin surfaces thus providing *mechanical* protection

Since from powders one expects mainly these *physical* effects their composition is usually inert Most frequently the dermatologist uses a mixture of zinc oxide and talcum

Rx Zinc oxide
Talcum purified āā 50.0

Formerly starch (amylum tritici or amylum oryzae) which is a finer powder was used instead of talcum It has been abandoned because dermatologists fear that organic powders may ferment and decompose and conceivably lead to irritations, especially in intertriginous areas

In theory mobile or sliding powders (e g lycopodium) whose microscopically uniform granules are covered with prominences and impregnated with fat are highly recommended These powders spread easily almost like a liquid Especially if used in a cooling paste they were supposed to have a more marked cooling effect because the loosely interspersed water could easily evaporate They have however failed to become popular in practice

and tars (except liquor carbonis detergens) in higher percentages are not miscible with lotions unless an emulsifier such as tincture of quillaja or tragacanth is added. Therefore, it is advisable that the physician become accustomed to and try to get by with a few standard prescriptions, instead of constantly experimenting with new combinations with which he has not had personal experience. If he does not stick to this rule he is likely to hear frequent complaints from druggists and patients.

Treatment with lotions is actually only a modified or improved version of treatment with powders. Fundamentally a lotion has essentially the same effect as a powder but this effect is a bit more pronounced because it leaves a thicker layer of powder. From this fact follow the *indications for lotion treatment*. Since depth effects from a thin layer of powder are small a lotion is suitable only for *superficial* dermatoses, especially for very *slightly oozing* conditions, for reasons which have already been discussed under powder treatment. The domain of the lotion is in the treatment of large surfaces, since it saves dressings and does not get sticky as ointments do. A disadvantage is that dried lotions release dust, which is annoying especially if dark clothes are worn by the patient. Lotion treatment, however has become so important because it can be used almost without risk in even very irritable dermatoses which are often made worse by salves (especially acute eczemas). Thus the lotion represents the modern dermatologist's typical starting treatment for acute and subacute eczemas. It is also indispensable in many cases showing initial intolerance of salves. Treatment with lotions is also *contraindicated* in the presence of marked oozing because the secretion is likely to cake the powder even more than it does with ordinary powder treatment. It is liable to form hard mortar-like masses and little crumbs, which irritate mechanically favor pyogenic infections, and start ulcerations. This danger can be reduced by alternating with wet applications. Frequently lotions dry the skin too much so that it becomes brittle and cracked especially in areas with a thick horny covering (palms). In such cases one has to increase the glycerin content of the lotion or change to pastes. Of course treatment with lotions is impossible in hairy areas because the glycerin-powder mixture causes the hairs *to stick together*. Lotion treatment may be quite unpleasant if applied to the scrotum because involuntary movements of the muscular coat of the scrotum may cause pulling on scrotal hairs which are fixed in the tough mass of the dried lotion. Finally lotions are useless in all more deep-seated processes unless they contain strong chemical agents.

The *application* of lotions is usually done with the fingers after a sufficient amount has been transferred from the bottle to the skin because the finger can spread it evenly in a thin layer. In irritable dermatoses it is paramount not to cleanse the skin each time but rather to paint over the old deposits as long as possible. If the lotion has to be removed this is most simply done with warm water or more easily with benzene or 30 per cent olive oil in benzene on satu

The original formula for a shake lotion calls for *equal parts (by weight) of powder and liquid*

Rx Zinc oxide
Talc
Glycerin pur
Water āā 25 0

This mixture however is rather thick and tends to dry too rapidly after spreading Therefore it is better to use only 40 per cent powder Twenty-five per cent glycerin makes the lotion somewhat sticky so 10–15 per cent is sufficient. If one replaces half the water with alcohol, the mixture dries more quickly Therefore, we use the following well known standard formula

Rx Zinc oxide
Talc āā 20
Glycerin 15
10% Grain alcohol
Water āā q.s. ad 100

Chemically active *drugs* may be added in small quantities, provided that they are sufficiently soluble (e.g 2 per cent ichthyol, tumenol or resorcin) If larger amounts of liquid drugs are to be added their quantity should diminish the amount of alcohol and water

Rx Ichthammol (ichthyol) 20
Zinc oxide
Talc āā 20
Glycerin 15
Alcohol
Water āā q.s. ad 100

However if the added medicaments are powders their weight must be deducted from the zinc oxide and talcum

Rx Sulfur precip 20
Zinc oxide
Talc āā 10
Glycerin 15
Alcohol
Water āā q.s. ad 100

In addition to these generalizations there are many special details to be considered which can only be outlined here It is important to keep in mind that many drugs even in small quantities change the consistency of a lotion. For example, 2 per cent resorcinol or 2 per cent ichthyol makes it thinner which may necessitate the addition of more powder Two per cent salicylic acid tends to make the lotion stiffer so that 50 per cent liquid becomes suitable If 2 per cent salicylic acid and 2 per cent resorcinol are combined the consistency of the lotion remains unchanged A special difficulty is caused by the fact that oils

grease component of the paste. Powder additives should accordingly reduce the inert powder of the paste

Rx Sulfur precip. 20
Zinc oxide
Talc āā 15
Petrolatum q.s. ad 100

Instead of solid greases, such as petrolatum vaseline, or lard, one may also use oils to make pastes. This results in an oil paste like *zinc oxide oil* which consists of equal parts of oil and zinc oxide

Rx Zinc oxide
Ol. sesami āā 50

Zinc oil is much softer than the zinc paste in the strict sense. Therefore it can be applied more easily even on sensitive skin and does not form rolls so easily On the other hand the oil does not make the skin supple it may after long use, even lead to troublesome dryness. The practical importance of zinc oxide oil is found in the fact that acute dermatitides which frequently do not tolerate vaseline, lanolin lard or other greases usually tolerate oil very well. Therefore zinc oxide oil is equivalent to lotions in such cases, and usually only experience will tell which of these two forms of application deserves preference.

Pasta zinci oleosa is a zinc oxide oil with 60 per cent zinc oxide instead of 50 per cent. This preparation of course is somewhat firmer Its consistency depends not only on the quantity of the added powder but also on the oil used and the method of preparation, especially the time spent in mixing and stirring All these factors make the quality of zinc oxide oil rather variable In addition its quality also depends much on the added medicaments. Sulfur makes it thinner so that if larger amounts of sulfur are to be incorporated, only a part of it must be deducted from the zinc oxide, e.g

Rx Sulfur precip.
Zinc oxide āā 30
Ol. sesami q.s. ad 100

Sometimes the influence of a medicament on the consistency is unpredictable For this reason it is not advisable to prescribe boric acid in zinc oxide oil because this mixture sometimes turns out solid and sometimes liquid. The reason for this behavior is not well understood.

The *effects* of pastes correspond to their composition Pastes are drying though they still keep the skin somewhat supple and soft. They may therefore, be used in moderately oozing conditions, especially the *hard* (stiff) pastes. Their powder component makes them stick better when applied thinly to the skin than greasy salves, which are promptly absorbed by clothing Thus they give better mechanical protection. Since this protective cover is not entirely

rated cotton swabs moved gently on the skin in circles. Alcohol is not suitable because it necessarily leads to too vigorous rubbing In general thorough removal of the lotion is difficult and not desirable A zinc oxide–oil mixture (see below) is easier to remove. If a lotion has dried out and become too thick it can often be restored by the gradual addition of warm water until the right consistency has been reached

PASTES—POWDER OINTMENTS

As the lotion in its original basic form consists of half powder and half liquid a paste consists of half powder and half grease The basic formula for zinc oxide paste therefore is as follows

Rx Zinc oxide
Talc āā 25
Petrolatum q.s. ad 100

Formerly starch was used instead of talc but this is no longer so popular with dermatologists because organic powders tend to decompose. Non-dermatologists however still frequently use the famous starch containing Lassar's paste which for unknown reasons also often contains 2 per cent salicylic acid

Zinc oxide paste according to the basic formula given here is rather dry and difficult to apply to sensitive skin without forming rolls of grease and powder which lie around in the bed and soil the floor Therefore zinc oxide paste is often prescribed as *soft zinc oxide paste* (pasta zinci mollis) by reducing the powder content to 40 per cent

Rx Zinc oxide
Talc āā 20
Petrolatum q.s ad 100

This soft paste is less drying keeps the skin more supple and has a little more depth effect. On the other hand it has the disadvantage that it is less well tolerated if the patient is intolerant of ointments. If this latter is not a matter of concern one may reduce the powder still further for instance by omitting the tal altogether One then has common zinc oxide ointment (unguentum Wilsoni) which has the following formula

Rx Zinc oxide 20
Petrolatum q.s ad 100

Lard instead of vaseline hardens a paste and makes it necessary to reduce the powder content still further

For the addition of chemically active ingredients, the same rules which apply to lotions also apply to pastes. No change in the basic paste formula is necessary if only small percentages are to be added If larger amounts of liquid medicaments (e g tars) are added their weight has to be subtracted from the

or similar substances. Some are used in their original form (especially on the hair) some are first given the consistency of pastes and salves by additions of various kinds (powders, diachylon)

By far the most commonly used ointment base is *petrolatum* (vaseline, petroleum jelly) Yellow petrolatum (petrolatum flavum) consists of butter soft paraffins which are residual products in the refining of petroleum. By bleaching or adsorption of colored impurities, white petrolatum (petrolatum album) is obtained, which some dermatologists believe to be better while others think that it irritates more frequently because of residual bleaching agents, especially sulfur dioxide, if such were used in its preparation. In any case no vaseline should contain any volatile components which can be detected by a petroleum odor Contamination with these and other residues from petroleum distillation may cause not only irritations but also comedones, folliculitides, pigmentations, keratoses, and even carcinomas, as was observed during World War I

The great advantage of petrolatum is that it keeps well and does not deteriorate by turning rancid, like fats. Even in a thin layer petrolatum keeps the skin supple to a particularly high degree It is less suitable for hairy areas because it causes the hairs to stick together and is also difficult to wash out with water and soap. Since its melting point lies close to skin temperature, it becomes so thin after application to warm parts of the body surface that it is easily absorbed by clothing Therefore it is sometimes used in mixtures with other greases, e.g lanolin.

Paraffin is used only seldom, in the form of unguentum paraffini which is a mixture of solid and liquid paraffins (mineral oil) melted together It also is not perishable and is supposed to be particularly non-irritating. Many practitioners believe that it does not spread so well on the skin as does petrolatum

Lard (axungia or adeps suillus) is easy to rub in and spread on the skin. This fat therefore is well suited for hairy parts, especially since it can be easily washed out with soap and water For a long time, it was the most commonly used ointment base but in more recent times it has been replaced by petrolatum because lard changes its consistency too much with the temperature and also turns rancid too rapidly The latter shortcoming cannot always be sufficiently prevented by the traditional addition of benzoin (adeps benzoinatus) which sometimes causes irritation. Yet, considering the frequent sensitivity of eczemas to petrolatum, one frequently has good reason to remember lard as an ointment base. If lard is not tolerated, one has to try oil glycerin, or lotions. Since lard becomes a thin liquid on the skin it is frequently mixed with other substances, e.g 15 per cent beeswax or 10 per cent zinc oxide

Another important ointment base is *lanolin* (adeps lanae, wool fat) Pure lanolin (anhydrous lanolin) represents such a tough mass that it is not suitable for an ointment. However it takes up water easily and then lubricates well.

impermeable, they do not have so deep an effect as do salves. The advantage of this weaker effect is that they rarely irritate even though this still occurs more often than with lotions.

Accordingly, their field of *application* is found essentially in the treatment of superficial inflammations of the skin including slightly oozing acute and especially chronic eczemas provided that they do not seem particularly irritable. In infiltrative processes they are used only as *paste dressings* because this type of administration is almost impermeable. Still deeper effects may be brought about by strong chemical additives (e g chrysarobin in the treatment of psoriasis or 20–40 per cent resorcinol to produce a peeling effect in acne)

The *contraindications* for pastes are essentially the same as those for lotions, namely marked oozing and also conditions in hairy areas. It is dangerous to use bland pastes for the treatment or aftercare of trivial pyogenic infections (impetigo furunculosis) since the pyogenic cocci continue to grow underneath the paste and then are simply disseminated by the repeated regular applications Unceasing recurrences from such autoinoculation are the usual sequel. In these cases disinfectant agents (sulfur vioform etc) must be added or disinfectant salves should be used

The *application* of pastes is mostly done by simply spreading them on with the fingers One may dust powder on top of the application to protect the clothing and to keep the paste from being absorbed by the clothing Pastes with staining ingredients (chrysarobin) are best applied with a toothbrush If an increased depth effect is desired one spreads the paste with a tongue depressor about ½–1 mm thick on a strong piece of material (flannel linen cotton but *not* gauze) and covers the diseased area of the skin with it To prevent shifting a thin layer of cotton is placed over it and the whole is fixed with a bandage. If the paste is merely spread on the skin the depth effect increases with the softness of the paste

As in the case of lotions it is wrong to remove the residues of pastes before each new treatment. The cleansing procedure may irritate and weaken the effect of the treatment Therefore in changing the paste dressings one should remove only the large particles which can easily be picked up One should never use water and soap for the removal of the paste since it would necessitate vigorous rubbing The most gentle procedure is dabbing with oily benzene or cautious wiping with olive oil the latter being especially suitable for oil pastes.

OINTMENTS

Ointments (salves) are greases or grease-resembling substances which at room temperature, have a butter like consistency They are used because of this consistency and not because of their chemical composition which may be quite varied Only some of them are true *fats* others are *hydrocarbons*

In a wider sense the ointments also include the oils which are liquid greases

or similar substances. Some are used in their original form (especially on the hair) some are first given the consistency of pastes and salves by additions of various kinds (powders, diachylon)

By far the most commonly used ointment base is *petrolatum* (vaseline petroleum jelly) Yellow petrolatum (petrolatum flavum) consists of butter soft paraffins which are residual products in the refining of petroleum. By bleaching or adsorption of colored impurities, white petrolatum (petrolatum album) is obtained which some dermatologists believe to be better while others think that it irritates more frequently because of residual bleaching agents, especially sulfur dioxide if such were used in its preparation. In any case, no vaseline should contain any volatile components which can be detected by a petroleum odor Contamination with these and other residues from petroleum distillation may cause not only irritations but also comedones fol liculitides, pigmentations, keratoses and even carcinomas, as was observed during World War I.

The great advantage of petrolatum is that it keeps well and does not de teriorate by turning rancid, like fats. Even in a thin layer petrolatum keeps the skin supple to a particularly high degree. It is less suitable for hairy areas be cause it causes the hairs to stick together and is also difficult to wash out with water and soap. Since its melting point lies close to skin temperature it becomes so thin after application to warm parts of the body surface that it is easily ab- sorbed by clothing Therefore it is sometimes used in mixtures with other greases, e.g. lanolin.

Paraffin is used only seldom in the form of unguentum paraffini, which is a mixture of solid and liquid paraffins (mineral oil) melted together It also is not perishable and is supposed to be particularly non-irritating Many practitioners believe that it does not spread so well on the skin as does petrolatum.

Lard (axungia or adeps suillus) is easy to rub in and spread on the skin. This fat, therefore is well suited for hairy parts, especially since it can be easily washed out with soap and water For a long time, it was the most commonly used ointment base, but in more recent times it has been replaced by petro- latum because lard changes its consistency too much with the temperature and also turns rancid too rapidly The latter shortcoming cannot always be suf ficiently prevented by the traditional addition of benzoin (adeps benzoinatus) which sometimes causes irritation. Yet, considering the frequent sensitivity of eczemas to petrolatum one frequently has good reason to remember lard as an ointment base. If lard is not tolerated one has to try oil glycerin or lotions. Since lard becomes a thin liquid on the skin, it is frequently mixed with other substances, e.g 15 per cent beeswax or 10 per cent zinc oxide.

Another important ointment base is *lanolin* (adeps lanae, wool fat) Pure lanolin (anhydrous lanolin) represents such a tough mass that it is not suitable for an ointment. However it takes up water easily and then lubricates well.

Because of its water content it should be classified with the cooling salves, which will be discussed later

While pure lanolin is too tough *oils* are too liquid to be used alone to any large extent. If applied to the skin, oils vanish quickly partly by drying and partly by being soaked up by the clothes. Nor are they particularly suitable for the scalp because they make the hair too greasy They are used with advantage in softening crusts and thick masses of scales in the hair (so-called oil cap) Oils are mainly used as emollients to soften other ointment bases and as additives to medicaments which would be too drying without oil (alcoholic hair tonics, benzene) They also find much use after having been given the consistency of pastes or ointments by the addition of other ingredients. Thus by mixing oil and powder in equal amounts we get the popular zinc oxide oil and by mixing oil and diachylon (lead plaster) in equal parts unguentum diachylon Hebrae is obtained which is a well tried particularly bland but also astringent and probably disinfectant ointment. Instead of oil petrolatum may be used for the preparation of unguentum diachylon.

The most frequently used oils are as follows

Oleum olivarum olive oil which has the reputation of being particularly non irritating though it readily becomes rancid

Oleum sesami *oleum rapae* (rapeseed oil) and *oleum arachidis* (peanut oil) are cheaper than olive oil and probably just as good

Oleum ricini (castor oil) is often added to alcoholic hair tonics It is also a customary solvent for salicylic acid

Oleum amygdalarum (oil of almonds) is colorless and odorless but expensive

Oleum lini (linseed oil) has a bad odor and a rather pronounced drying effect Despite these disadvantages it is mixed with equal parts of limewater and used for extensive erosions and painful sunburns (burn liniment)

Oleum jecoris aselli (cod liver oil) from fish livers has a particularly macerating effect and a repulsive smell Yet mixed with thicker ointments it enjoys a special reputation in the treatment of chronic ulcers and tuberculids, probably less on empirical grounds than because of special expectations based on its vitamin D_3 content.

Ointment bases can be hardened by the addition of tough substances so that it is possible to cast them into molds. Such ointment sticks or pencils (e.g 30 per cent wax in lanolin) are commonly used cosmetically as lipsticks. Occasionally they find therapeutic use (e.g in the treatment of chapped lips)

The *indications* for ointments are very numerous. They serve to relieve dryness and brittleness and the fissures which accompany these states. Furthermore they are used for the gentle removal of squamous and crusty deposits (chronic eczemas psoriasis, impetigo ulcers) They can also be used in hairy areas where however, oils are more suitable. Ointment dressings may on occasion provide a protective covering for oozing processes if because of the pro-

fuse secretion, drying by lotions and pastes is no longer possible and most dressings macerate too much. They are especially indicated as protective coverings for suppurating conditions which are too deeply seated to be reached by the effects of paste dressings. A thick, soft layer of ointment not only provides an excellent mechanical protection but also prevents drying out and stasis of secretions which might be caused by dry dressings and possibly also by paste dressings.

The salves also play a most important role as *vehicles of chemically active agents*. Such mixtures in some cases are officially recognized (5 per cent ammoniated mercury ointment, U.S.P.)

Much more important in practice are compounds which the physician may apply in countless modifications according to the requirements of the case and guided by detailed knowledge. The most simple method is to add to the salves either powders (customarily 10 per cent) or of course all drugs which are soluble in fats and oils. The solubility of a drug varies according to the ointment base used. For instance salicylic acid dissolves more easily in oils than in petrolatum. Some chemicals must be combined with others in order to become soluble. For example, iodine is soluble in petrolatum if potassium iodide is added. Drugs which are not soluble must be finely powdered and evenly dispersed. Agents containing water can ordinarily be added in small quantities only since higher concentrations separate quickly (laking). It is very important to know that some medicaments are more easily taken up from one ointment base than from others (salicylic acid, taken up better from Aquaphor than from lard) and that some have a much stronger effect in one base than in another (chrysarobin acts more strongly in petrolatum than in lanolin). Some medicaments are generally weak in ointments and must first be applied to the skin and then covered with an ointment dressing to obtain a good depth effect.

In irritable dermatoses, fats and similar substances very frequently cause irritation. This is the reason for the most important *contraindication* to treatment with ointments namely most acute inflammatory and especially oozing dermatoses. In these cases it is better to start treatment with compresses, lotions, and pastes or at most, cooling salves. Treatment with ointments is the method of choice in chronic, scaling and deep processes. In order to enhance further the effect of ointments in squamous and keratotic processes, one may add 5–10–20 per cent salicylic acid which dissolves the horny layer and opens the way for the penetration of other medicaments.

The *application* of ointments is done either directly or with dressings. Salves for direct application should not be too thin, otherwise they will be too rapidly soaked up by clothing. If there is not too much dryness and scaling one may add a little powder or sprinkle powder lightly on the applied salve. Ointments are particularly easily absorbed by linen and cotton. In this respect, wool would be better but it is too warm and also scratchy which frequently leads

to irritation A thin and cheap cotton material next to the anointed skin is most suitable in practice

For *ointment dressings* the salve is not spread directly on the skin but in a 1 mm thick layer on a piece of rather firm material Very suitable for this purpose is the smooth side of linen flannel or cotton Old thin cotton material (e.g many times laundered shirts or bed sheets) is excellent because it stays snugly on the skin and does not shift out of place. If the material is stiffer one has to make some radial cuts after the salve has been spread on which will prevent folds especially on curved parts. Gauze would fit better but is not suitable because the ointment penetrates through it The pieces used should not exceed hand size since several small pieces which overlap in shingle fashion make for better fit than does one large piece To prevent shifting still further a thin layer of cotton (tailor s padding two layers with the glued sides touching inside) is placed on top The cotton layer is then fixed by bandages. Since a good fit of the bandage is of extreme importance one makes use of certain special techniques for different parts of the body For the head one uses bathing caps or oversize baseball caps for the face masks cut to size with ribbons on forehead and chin for the ears ear muffs for hands washable white cotton gloves for the feet old stockings for the scrotum a suspensory for the penis a double stockinette type tubular bandage for the anus a T bandage Of great practical value are stocking like tubular bandages of thin cotton material (*stockinette*) They come in five sizes suitable for fingers arms, legs thighs and head. They are cut to double the length of the area to be covered and then applied like a stocking The free length is twisted once and then pulled back over the extremity which is already covered with one layer Thus a sock sleeve mitten mask or cap can easily be improvised to keep the ointment dressing in place. All salve dressings are changed twice daily after gentle and only superficial cleansing The least irritating cleansing agents are benzene and oil. The surroundings of ulcers may frequently become macerated or irritated by some ointments. If such is the case, the surrounding areas should be covered with zinc oxide paste lotion or one of the modern silicone ointments before the salve dressing is applied

COOLING OINTMENTS—WATER OINTMENTS

Some ointment bases have the ability to absorb water in relatively large amounts. This is of therapeutic importance because such ointments are also able to absorb serum in oozing dermatoses and to cause a certain amount of cooling by evaporation of absorbed water This cooling by evaporation has an antiphlogistic effect while ordinary ointments easily cause overheating Therefore the water-containing salves are called *cold creams* or *cooling ointments* The classical base for this type of ointment is *lanolin* (adeps lanae wool fat) This material in some respects is similar to human sebum It may however irritate sensitive skin and it has a somewhat disagreeable odor Anhydrous or refined

wool fat (adeps lanae or lanolin U.S.P.) which is very thick and therefore unfit for ointment use absorbs three times its own weight of water i.e. up to 300 per cent while vaseline absorbs only 8 per cent at the most. Mixed with a sufficient amount of water lanolin is a suitable ointment. Ordinary commercial lanolin (hydrous wool fat, adeps lanae hydrosus U.S.P.) is such a mixture of lanolin with 27 per cent water

The cooling effect of water-containing salves depends by no means only on their water content but also on the rate of evaporation of this water. In lanolin ointments, for instance the lanolin surrounds the water droplets very closely. Therefore, their cooling effects are not too pronounced. Other water-containing salves may dry too fast in the jar or they may be excessively influenced by temperature, becoming too soft in the summertime. For all these reasons, the water-containing fats are usually mixed with other bases, e.g. lanolin with petrolatum. The basic formula for a lanolin cooling ointment would thus be as follows

Rx	Petrolatum	20
	Aquae	
	Lanolin anhydr. āā q.s. ad	100

Instead of pure water medicated especially astringent, liquids like boric acid solution or Burow's solution (aluminum acetate solution liquor alumini acetatis U.S.P. ½–2 per cent) are popularly used because it is assumed that such agents enhance the effect of the salve.

Instead of petrolatum sometimes lard oil, or glycerin or combinations of such bases (e.g. 20 per cent oil 20 per cent glycerin and lanolin petrolatum āā) are employed to improve the consistency of cooling salves. This leads to complicated formulas, with the drawback that, in the case of irritation one does not know which of the ingredients to blame. The addition of medicaments to such formulas also creates surprising problems of miscibility chemical compatibility tolerance and therapeutic effect. This is the reason why such complex ointment mixtures as official rose-water ointment (unguentum aquae rosae U.S.P.) which is composed of wax, spermacetum oil of sweet almonds, and water and which still can absorb 25 per cent of water does not play the role which one would expect theoretically. The same is true of numerous proprietary cold creams. However we have in *Aquaphor* a cooling ointment consisting almost exclusively of petrolatum which by a small addition of lanolin derivatives, has been rendered as water-miscible as pure lanolin. Of course it has the short coming of the pure petrolatum, which often is not well tolerated in acute skin diseases.

There also exists another shortcoming in the cooling salves which have been named so far and this is that their cooling effect is restricted because their water droplets are sealed in grease and therefore are unable to evaporate (water-in-oil salves). One really would like a cooling ointment in which the oil drops

were suspended in the liquid Such oil in water salves which have been prepared also have the advantage of not leaving a greasy sheen They seem to vanish into the skin (vanishing cream) However they dry quickly and therefore do not keep well in the jar Also their preparation is a rather complicated procedure because it is often necessary to make use of emulsifying agents in order to keep the oil droplets from coalescing These ointments also sometimes decompose or harden in an unpredictable way as soon as chemically active medicaments are added Therefore these theoretically well founded ointment bases have not been able to replace the simple lotions and oil pastes in the treatment of irritable dermatoses.

As an example of a vanishing cream I would like to mention *Lanette wax ointment* It consists of 10–20 per cent Lanette wax N (mainly cetyl alcohol) and an equal amount of oil in water

Rx	Cerae Lanette N	
	Ol. sesami āā	15 0
	Aqua q.s. ad	100 00

For cosmetic purposes one adds spermacetum and wax (3–5 per cent) in order to give the salve more smoothness To insure good consistency one should make sure that the solid and liquid fats are used in approximately equal amounts. One should also add a disinfectant (2 per cent salicylic acid) to keep the ointment from getting moldy Its preparation depends very much on the skill of the pharmacist If the ointment is cooled too fast it will become granular so that it has to be reheated The water must be added very slowly drop by drop Drugs are added later A prescription of this kind reads about as follows

Rx	Cerae Lanette N	
	Ol sesami āā	15 0
	Aqua ad	100 0
	Ammoniated mercury	
	Liq carbon deterg āā	10 0

The later addition of medicaments makes the prescribing difficult since it is important not to lose sight of the percentages of the added ingredients.

A special position among ointment bases is occupied by *glycerin* (glycerinum U S P glycerol—a by product of soap manufacturing) because it is often tolerated when sensitivity to ointments exists It should however not be used undiluted since pure glycerin is hygroscopic and therefore causes burning and irritates the skin. It should always contain at least 20 per cent water Its importance in the preparation of lotions has already been mentioned It can also be prepared as a salve by boiling it with 6 per cent starch and 20 per cent water a procedure which results in unguentum glycerini

Of course glycerin may also irritate in some cases though this occurs less frequently than with petrolatum and fats It is also somewhat sticky However

glycerin ointment is a valuable help when salves and lotions are not tolerated or when lotions and pastes are too drying or too conspicuous on the face.

COOLING PASTES—WATER PASTES

By the addition of sufficient quantities of inert powders, cooling salves are converted into *cooling pastes*. Thus one may add 20 per cent zinc oxide *to a* cooling salve with aluminum aceticum

Rx	Zinc oxide	
	Petrolatum āā	20
	Lanolin anhydr	
	Sol. alumin acetic. (2 per cent) āā q s. ad	100 0

Or one may prepare an oil-in water cooling paste by adding powder to the Lanette wax ointment

Rx	Cerae Lanette N	
	Ol. arachid. āā	10
	Zinc oxide	20
	Water q s ad	100

By adding liquid to zinc oxide oils, a corresponding result may be obtained. Such a cooling zinc oxide oil consisting of equal parts of linseed oil limewater zinc oxide and chalk had already been recommended by Unna. A similar very simple prescription which has become more popular consists of equal parts of olive oil limewater and zinc oxide (Polano) It has an effect similar to that of ordinary zinc oxide oil. If one wants to prescribe a liquid mixture with an *acid reaction*, one may use, for example, sol alumin. acetici instead of the limewater but then one has to add an emulsifier e.g 2 drops of oleinic acid and 2 drops of 30 *per cent* potassium hydroxide for every 10 gm. of oil.

Polano s zinc oxide oil is a water-in-oil paste. If one desires to prescribe an oil-in-water paste to obtain a more marked cooling effect one needs only to decrease the oil and to increase the other liquids in a proportion of 1 part oil 2 parts zinc oxide, and 3 parts limewater Of course this type of zinc oxide oil has a greater drying effect on the skin.

The *indications* for cooling ointment and cooling pastes can easily be derived from the preceding discussion Because of their cooling effect, they are suitable for acute inflammatory skin diseases. However in general, they cannot compete with lotions and zinc oxide oils because their cooling effects vary widely according to their composition and also because by the mixture of a large number of different fats and liquids, including emulsifiers, the likelihood of primary irritations increases substantially If such an irritation then occurs, one never knows by which ingredient it was caused. It then becomes necessary to start so to speak, over again with a completely different ointment base or risk the repetition of the irritation. My opinion with regard to the therapeutic effects of chemicals added to cooling salves is just as guarded The preparation alone of

such ointment mixtures frequently causes the greatest difficulties owing to their complicated and changing composition which makes it difficult or impossible to find out how they compare with ordinary petrolatum salves Also with ordinary salves it is generally easily possible to increase methodically the concentrations of various active ingredients or to combine them to get desired effects This cannot be done along the same lines with the cooling ointments.

Cooling ointments seem most suitable for non irritable dry dermatoses because they are not greasy enough to cause glossiness on exposed areas (vanishing cream) and they do not soil the underwear However for the same reasons, they fail to make the skin sufficiently supple and since they form a more penetrable covering they also fail to make the incorporated ingredients work at their *maximal* power In the case of strong medicaments which stain the skin and underwear the agreeable cosmetic-cream quality of cooling salves no longer has a great advantage since the strong salves are more annoying by their staining power than by their greasiness.

However great the significance of the cooling salves may be in cosmetics, their usefulness in the treatment of skin diseases is limited. Ointment bases which are best *cosmetically* are by no means best *therapeutically*

The relative *contraindications* of the cooling ointments and pastes follow from their disadvantages in acute and oozing dermatoses they irritate more frequently than do lotions and zinc oxide oils. In chronic and scaling skin diseases they are not greasy enough and therefore are too weak even with added chemicals It is also annoying that they are quite perishable which makes their constant replacement necessary if one does not want to find dried out and moldy salves in the jars

VARNISHES AND PLASTERS

Besides the vehicles consisting of liquids powders and greases which have already been discussed there are *other substances* which are used as vehicles for chemically effective medicaments.

Among these are the *varnishes* These are liquids used for the solution or suspension of medicaments which after having been brushed or painted on the skin dry quickly and leave a solid covering It is possible to wash cautiously over such varnishes containing water insoluble organic substances without removing them Most medicaments are more effective in varnishes than in an ordinary solution (e g bichloride of mercury acts more strongly when dissolved in collodion than in alcohol) Accordingly they also irritate more frequently Obviously this is caused by the stronger depth effect produced by the impermeable covering On the other hand there are medicaments such as chrysarobin which are more likely to irritate in a varnish but exhibit only a weaker therapeutic effect than for example in zinc paste. I am at a loss to explain this behavior It is an advantage of treatment with varnishes that they can be used more easily on skin areas where dressings are difficult to apply or

are uncomfortable. They also scarcely soil the underwear because they do not release staining drugs so readily as do greasy salves and pastes.

The varnishes most frequently used by dermatologists are tincture of benzoin (tinctura benzoini U.S.P.) traumaticin, and collodion. *Benzoin tincture* is a 10 per cent solution of benzoin resin in alcohol. It is used to dissolve tar preparations (tumenol and anthrarobin in Arning's tincture) and also corrosive sublimate. Benzoin tincture forms only a loose crumbly covering.

Traumaticin is a 10 per cent solution of guttapercha in chloroform. It is generally recommended as a vehicle for chrysarobin in the treatment of psoriasis but it frequently causes irritation. It forms a somewhat more coherent and flexible covering, which, however also quickly rubs off.

A firmly adherent, durable membrane is obtained with flexible collodion, a thick solution of nitrocellulose in equal parts of alcohol and ether with an addition of per cent castor oil for increased flexibility. Such collodion is used mainly as a solvent for salicylic acid to soften horny masses (calluses, corns) but may also be used for chrysarobin and pyrogallic acid in the treatment of psoriasis.

Varnish treatment is *indicated* in eruptions on exposed areas where dressings and plasters are too conspicuous or over joints where they do not stay well. They are also useful in protecting clothing in scattered eruptions such as a few lesions over the trunk and extremities. They are also very practical in those lesions where the drug must be exactly localized to prevent irritation of surrounding normal skin (e.g. salicylic acid in calluses). They are useless and therefore *contraindicated* on eroded and ulcerated areas because they do not cling to oozing skin. They should not be used in hairy areas because they glue the hairs together.

Before *applying* the varnishes, one should make sure that the skin is dry. It is not desirable to remove the residues of previous applications. One paints right over the old coat of varnish, just as is done with lotions.

Related to the common varnishes are the *gelatin varnishes*. These are coverings which are liquid at higher temperatures and therefore must be applied hot. On cooling they solidify and shrink. They contain glue, gelatin, or tragacanth as a binder. The most frequently used such preparation is the zinc gelatin dressing (Unna's boot) used in the treatment of leg ulcers. In this case the shrinking of the cooling gelatin helps compress the varicose veins of the lower leg. Here might also be mentioned those ointments which have no other purpose than to form a protective covering and which, therefore might be called *ointment varnishes*. Their purpose may be to afford protection from contact with chemical agents (occupational protective creams) or protection from rays (light protective creams). The occupational protective creams mostly contain waxes or even actual varnishes in order to make them harder.[2] In some instances

2. More recently silicone-containing protective ointments have largely replaced the older types without clear proof of superiority.

they must also prevent soiling of the product which the worker is handling, which requires that they cling firmly to the skin The sun protective creams are usually ordinary petrolatum or cooling salves which contain ultraviolet filtering substances (e g , tannic acid para aminobenzoic acid and its esters opaque powders) Of course it is much cleaner and more practical to use aqueous or alcoholic solutions for protection against sun rays e.g 5 per cent para-aminobenzoic acid

Finally we have to discuss the *plasters* These are medicaments which are spread on fabric and possess adhesive power The adhesion is accomplished by the addition of resins (rosin resin of dammar) which irritate the skin so frequently that generally only small quantities of them can be used Therefore, medicated plasters usually do not stick well and have to be attached with adhesive tape Because plasters form an impermeable covering they have a strong depth effect For this reason they are popularly used as salicylic acid-soap plasters to soften horny masses and as phenol mercury plasters for the treatment of infiltrated boils. Plasters with tar chrysarobin or pyrogallic acid are well suited for the treatment of stubborn patches of psoriasis It is also a very practical technique to cover with overlapping strips of adhesive tape any lesions which have been painted with soiling solutions or varnishes. This not only protects the underwear but also increases the depth effect of the medication by shutting off the air and increasing the maceration On the other hand of course the increased effect also carries an increased danger of irritation Therefore this method of treatment is suitable only for very chronic cases.

The *indications* for plaster treatment essentially coincide with those for varnishes However medicaments have to be closely localized to a certain area because they would otherwise corrode the surrounding skin (e g salicylic acid on corns) Varnishes are more useful because plasters may shift out of place A special application of the plasters is their use in the removal of hair The old pitch plaster cap which once used to be pasted to the hairs in order to permit tearing them out with a mighty jerk after the tar had solidified has only historical interest because of the cruelty of the method It makes us appreciate the present bad times in comparison with the good old times. However it is practical and with good technique almost painless to remove hairs which have been loosened by X ray treatment by applying strips of adhesive tape and then with a sudden jerk peeling the strips off with the hairs sticking to them

Before *applying* plasters the skin has to be degreased with benzene or ether in order to make the plaster stick The hairs must be carefully removed otherwise the removal of the plaster is painful Even so the removal of a well attached plaster remains an unpleasant procedure unless it is done with a quick jerk in a peeling manner parallel to the skin without trying to pull the plaster off perpendicularly To remove a plaster most gently one should loosen it with benzene

CHAPTER NINE

Medications in the Stricter Sense (Active Ingredients)

Owing to their mechanical and physical effects, treatment with vehicles alone is of great importance. However in external treatment we often cannot do entirely without *chemotherapy* i.e. we have to add chemically active and effective *medicaments* Thus complete dermatologic prescriptions consist of the various vehicles used plus the chemotherapeutically active ingredients.

NOTE ON THE MANNER IN WHICH PRESCRIPTIONS ARE GIVEN IN THIS BOOK

It is customary for textbooks to present prescriptions in the same manner as they are prescribed for the pharmacist namely in the presumably necessary total amounts. For example, for a facial eruption this would be 60 gm. of ointment for the treatment of the whole body surface or for a hair tonic 200 gm. This has the great disadvantage that the formulas of complicated compounds frequently appear with different numbers and therefore are difficult to remember It is more important that this method prevents the physician from acquiring a distinct feeling for the strength of his prescription because he always first has to convert the prescribed absolute amounts of the effective medicaments into percentages (relative amounts) if he wants to compare them with other prescriptions. This is inconvenient, and even worse, it actually leads to a superficial, routine type of work. The dermatologist must always know how strong his treatment is, and therefore he must have at his fingertips and know by heart prescriptions which *indicate* this strength Thus it becomes the requirement of conscientious dermatologic therapy that in the textbook as well as in the notebook of the practitioner all prescriptions be figured in 100-gm total amounts so that all medications appear in percentages.

Therefore in the interest of better understanding of external therapy I have avoided the artificial difficulty which the textbooks of dermatology customarily have placed in their own way by the use of varying total amounts and have given all prescriptions in percentages. This results in another convenience,

namely that one can write all the ingredients one after the other and not below each other Thus the following prescription

Rx	Salicyl acid	6
	Ichthyol	3
	Zinc oxid	12
	Lanolin	
	Petrolatum āā q.s ad	60

would read

10% salicyl 5% ichthyol, 20% zinc oxide, lanol. petrolatum.

Since all ingredients are given in percentages anyway it is superfluous to repeat the per cent signs over and over again so I will henceforth omit them in prescriptions. Thus the above prescription would simply read

10 salicyl 5 ichthyol, 20 zinc oxide lan.-petrolat.

It is practical to keep the ingredients in a certain order to help prevent errors in reading I always start with the coloring matter then the often used salicylic acid ranging from inconsequential placebo amounts over keratoplastic to keratolytic quantities and if necessary antipruritics and finally the main medicaments and adjuncts. Of course there are other ways. All that matters is to adopt a method of prescribing which in a most simple manner immediately shows the composition and strength of each prescription I like to think that in ten or perhaps fifty years this method of prescribing will be generally adopted

ANTIECZEMATICS

Active medications may serve many different purposes. However in the foreground of dermatologic interest are those drugs which have an *anti inflammatory* effect the so-called *antieczematics* and *antipsoriatics* These are the drugs most frequently used in external treatment and their administration requires special knowledge and experience There are numerous theories and hypotheses about the basis for the favorable effects of antieczematics on epidermal and epidermo-cutaneous inflammations but they are of little avail in practical treatment Of real importance however is the expected *probability of help* from a particular remedy in a given case and also the likelihood of irritation and impairment which it carries It is on these empirically ascertained grounds that our practical therapy is founded and this is the way it should be

Knowledge about antieczematics starts with the fact that their effects are of *different strength* It is possible to arrange these medications in an order (scale) which starts with the mild drugs and progresses to the stronger and strongest ones. This fact is important because one must, of course in all changing and irritable ailments choose the mildest medications in the beginning and gradually if tolerance has been established make use of the stronger ones. On the other hand in chronic hardly changing inflammations, one must start right away with the stronger agents in order not to waste time with the mild

remedies. Naturally the strengths of the remedies depend partly on their chemical natures and partly on their concentrations. This means that there are drugs which, in low concentration, are mild antieczematics and in higher concentrations, are strong ones. In general, the position of a medicament in the scale is rather clearly defined.

Boric acid, salicylic acid and resorcinol (resorcin) are representatives of what are commonly believed to be antieczematics. *Boric acid (2 per cent)* so to speak is used ritually especially for compresses and in salves. The reason is not quite understandable. It is a most insignificant, mild astringent and disinfectant. It is presumably used in salves to prevent their spoiling. This hardly seems necessary since the customary boric acid ointment consists of petrolatum which does not spoil anyway. Therefore, the prescribing of boric acid seems to be completely unnecessary, the more so since it has *not* been demonstrated that it actually has antieczematic value. However it can be assumed that *salicylic acid* in weak concentrations of 0.1–2 per cent, has a keratoplastic effect, i.e., exerts a favorable influence on the new formation of the keratinous layer. Therefore it is suitable for oozing and acute eczemas. But this alone is not reason enough to add it routinely to all kinds of solutions for wet dressings, hair tonics, pastes, and ointments, as if there were no other way. In high concentrations (5–20 per cent) salicylic acid acts as a keratolytic. Therefore, in such concentrations it is the most important additive to strong antieczematics in the treatment of heavily scaly and keratotic dermatoses and where intensification of a caustic type of action is intended. It is not known whether salicylic acid alone is an antieczematic, but this is likely because of the apparently favorable effect of strong salicylic acid ointments on some squamous eczemas.

Resorcinol (metadihydroxybenzene) is also keratolytic and caustic if used in high concentrations. In weak concentrations (0.1–2 per cent) used in compresses, lotions, and pastes, it is an important remedy at the start of the treatment of eczemas. This is frequently explained by its partly astringent and partly reductive effects. I have, however, never been able to understand why this reductive quality should explain its effect on inflammatory processes, especially since some superficial inflammations of the skin, such as psoriasis, are hardly affected by resorcinol.

Sulfur is akin to resorcinol as far as effect and indications are concerned. It is used as a fine powder (sulfur precipitatum). Though it is employed in higher concentrations to intensify peeling as well as for a disinfectant purpose (pyodermas, trichophytons, scabies), it is at the same time a mild antieczematic which may be used, starting at 10 per cent. However, it must be kept in mind that a considerable number of people are sensitive to sulfur, at least in Holland. Sulfur dermatitis frequently has a *craquelé* appearance and often appears as a distant irritation in untreated areas. Sulfur is frequently used in lotions, pastes, and ointments, but it is of little use in water and hair tonics, since it is insol

uble. Proprietary soluble sulfur preparations have not yet become popular. Sulfur is considered very suitable in the treatment of erythemato-squamous (especially so-called seborrheic and intertriginous) eczemas. By the formation of hydrogen sulfide, especially in intertriginous areas, sulfur causes a disagreeable, though weak, odor of rotten eggs.

The transition from sulfur to the tars is formed by *ichthammol* (ichthyol), a thick black liquid distillate from bituminous shales. If mixed with zinc oxide in lotions, pastes and ointments, its odor is weak, and it stings but little. Because it is water-soluble, the stains come out in the laundry. It is most suitable as a transitional medication between bland and tar treatments. Since the addition of 2–3 per cent ichthammol colors lotions, pastes and zinc ointments a light tan (beige), it removes the conspicuous dead white color of such preparations and thus becomes an important help in the ambulatory treatment of the face. By further adding one half of 1 per cent of cinnabar, one may make such pastes and zinc ointments fairly skin-colored. Two to 20 per cent ichthammol N.F. very rarely irritates. Some *mercury* compounds are weak, yet in some cases very effective antieczematics. The most commonly used such compounds are ammoniated mercury (hydrargyrum ammoniatum U.S.P., white precipitate of mercury, 10–20 per cent) and yellow mercuric oxide U.S.P. (hydrargyrum oxydatum flavum U.S.P., 1 per cent, 2–5 per cent), the latter being almost exclusively used for eyelid salves. In using any mercury compound, of course, it is always necessary to remember that allergy to the metal is fairly common.

Together with the weaker drugs on the scale of antieczematics may be grouped a number of medicaments which are also used for the treatment of trivial inflammations, though it is not sure whether they have any antieczematic effect at all. For instance, there is *tannic acid* (2–5 per cent), an astringent, and medicinal *scarlet red* (2 per cent) (rubrum scarlatinum N.F.), a stimulant for poorly granulating wounds. Both agents, especially the latter, only exceptionally exhibit antieczematic activity. Subnitrate and subgallate of *bismuth* (10 per cent) are astringents. *Benzoin* (10 per cent) and many other preparations are used in the treatment of eczema, though nothing certain about their healing value is known.

Much better known and more reliable is the efficacy of the *stronger medications* on our scale, the true antieczematics in a stricter sense. Without exception they are tars or preparations containing or related to phenol. We have already mentioned resorcinol. Pure *phenol* U.S.P. (carbolic acid) is little used for its antieczematic effect because it may cause gangrene if used in aqueous solution for compresses on denuded areas. It is, however, well recognized in concentration of 2 per cent or less in alcohol lotions, and pastes as an antipruritic.

Most important in this group are the *tar preparations*. Tars are complex substances which are derived from wood or coal by dry distillation. They consist mainly of phenols and aromatic hydrocarbons. They constrict blood vessels

paralyze nerve endings and have antibiotic effects, but, most important, experience has proved that they are antieczematics. They irritate occasionally and therefore they are contraindicated in the routine treatment of acute eczemas. In all chronic eczemas, however they are the first choice. If used in very high concentrations over large areas, they may cause systemic poisoning with dark brown urine, albuminuria and coma, but this danger is generally overrated They must be used on the face cautiously because some of them sensitize the skin to light After protracted use they may cause a painless chronic folliculitis (tar acne) and also keratoses. In animal experiments they even provoke cancers, but for all practical purposes, this does not happen in therapeutic use

The most commonly used *wood tar* is rectified *birch tar* oil (oleum rusci oleum betulae empyreumaticum rectificatum N F 2 20 per cent or also pure) even though it has a strong and disagreeable odor It is an ingredient of the classical *Wilkinson's ointment* for which several formulas are known and which besides its use for eczema and psoriasis, was formerly used to treat scabies. It contains not only tar but also sulfur and soap (e g 10 ol. rusci 20 sulf precip., 30 green soap in petrolat.) Still stronger is the effect of *beech tar* (oleum fagi) and conifer tar (pix liquida) In patch tests, even on normal skin the latter usually elicits an erythema. If a milder tar effect is desired one may prescribe *juniper tar* (pix juniperi N F., cade oil oleum juniperis empyreumaticum) which stains less but still has an objectionable odor Without doubt, the best antieczematic tar is *coal tar* (pix carbonis U.S.P) It is used in pastes, in ointments, in chloroform, acetone and benzol solutions (tinctures) and also pure. It regularly causes folliculitis if used vigorously If one wants a mild coal tar effect *liquor carbonis detergens* (coal tar solution, liquor picis carbonis N.F 5–10–20 per cent to pure) a solution of coal tar in tincture of quillaja is suitable It may also be used on the face because it stains little and does not cause photosensitization.

There are of course, many other tar derivatives on the market e.g beta naphthol, which, however is unpopular because of its toxicity In this group too it is good advice to stick to a few preparations in order to get firsthand experience of the remedies one is working with. It is a drawback of tar treatment that the quality of the preparations may vary with the source of supply This may be sufficient reason to *give preference* to proprietary preparations over natural products. Unfortunately one cannot completely rely on the constant quality of all proprietary products either

The *strongest agents* for the treatment of superficial inflammations of the skin are pyrogallic acid (pyrogallol N F) and chrysarobin (chrysarobinum U S.P) Since these medicaments invariably cause *irritation* if they are applied vigorously their main indication is the *characteristically* non-irritable psoriasis. Therefore they should better be designated as *antipsoriatics* than as antieczematics. Yet it is difficult to do without them in the treatment of very chronic eczemas.

Pyrogallol N.F. (pyrogallic acid, trihydroxybenzol, 2–20 per cent) is a close relative of phenol. Not only does it act as an antieczematic and antipsoriatic but it is also a vigorous disinfectant. If used on erosions and ulcers, it has a strong caustic effect. If denuded skin areas of larger size are treated, systemic poisoning may occur. However, on intact skin the drug is innocuous in this respect. Extensive application of 10 per cent salves and tinctures over 8–10 days quite often results in a painful pustular irritation which heals only slowly under treatment with zinc oxide oil. Though freshly compounded pyrogallic acid petrolatum looks almost colorless, it stains the treated skin, the fingers, and the clothing black. Therefore, it is necessary to apply it with a tongue blade or toothbrush. Light-colored hair is stained gray to black and should therefore not be treated with pyrogallic acid. It may, however, be used on persons with dark hair, but the pillow cases and hats have to be protected. Pyrogallic acid is used mainly in salves. For residual psoriatic lesions and resistant verrucous lichen planus I like to apply it in collodion, though this method may cause violent and stubborn irritations. Because of its staining power, it cannot be used on the face and hands except in the hospital.

Less staining but also much weaker is *Lenigallol* (pyrogallol triacetate). In psoriasis it is usually ineffective. Occasionally, it is useful as an astringent antieczematic in vesicular hand eruptions (5 per cent in zinc oxide paste). In earlier day caustic treatment of lupus vulgaris, it proved to be almost as effective as pyrogallic acid and was far superior because it was painless and non-toxic.

The most effective antipsoriatic is *chrysarobin*. This is a brownish powder which is derived from crypts in the bark of a certain tree native of South America. It consists mainly of the anthranols of chrysophanic acid. The Indians knew its strong effect and used it in the treatment of fungus diseases of the skin. Its reducing property does not explain its outstanding antipsoriatic qualities, since stronger reducing agents, such as resorcinol, accomplish much less in this respect. If applied to the skin in increasing concentrations (0.1–20 per cent in zinc oxide paste, ointment, or varnish), chrysarobin sooner or later causes an explosive inflammation, which appears suddenly but just as suddenly vanishes if covered with a zinc oxide shake lotion. Greasy ointments make irritations from chrysarobin worse. In some cases, especially in acute and eruptive psoriasis, the irritation may cause an independently progressive dermatitis which may turn into a new psoriatic eruption and may even lead to a severe febrile universal erythroderma. Therefore, it is necessary to start with weak ointments, but one must increase the concentration regularly because the skin rapidly becomes accustomed to the medication and then the treatment becomes ineffective. If chrysarobin gets into the eyes, a very annoying conjunctivitis results, which in some cases may even lead to corneal ulceration if the treatment is not discontinued immediately. Therefore, the eyes must be protected. The patient should wear washable gloves and at night protective

goggles. This illustrates that the methodical execution of an extensive chrysarobin course of treatment needs circumspection and experience and virtually can be done only in a hospital, the more so because chrysarobin stains skin and underwear purplish brown. If strong concentrations are applied all articles of daily use which the patient may touch are likely to become stained.

Correct chrysarobin treatment requires special preparations. Besides gloves and goggles, there must be shirts and trousers which can be tied with ribbons on wrists and ankles and also special usually brown bed sheets and pillow cases, which have to be laundered separately from the other hospital linen. Chrysarobin must not be used on the face because of the danger to the eyes or on the scalp in light-haired persons because it stains the hair brownish purple.

One can read in many textbooks that chrysarobin is liable to attack the kidneys, causing albuminuria. This is not true. One is more likely to encounter kidney irritations from salicylic acid ointments and, exceptionally, from tar especially in children if the entire body is treated. Chrysarobin is not a kidney poison but rather a nerve poison. This is known only from animal experiments since in practice it has proved to be harmless for all internal organs. I have never seen any symptoms of internal poisoning though I have for decades used 20 per cent chrysarobin paste regularly on most regions of the body surface.

Anthralin (cignolin dihydroxy-anthranol) is more expensive than chrysarobin. Chemically it is but little different from the main component of chrysarobin and it has about the same advantages and disadvantages. Since it is generally somewhat stronger it can be used in lower concentrations, which makes it a little more adaptable for ambulatory treatment. There are persons who are hypersensitive to chrysarobin but not to anthralin and vice versa. Therefore one may make use of anthralin in some cases and of chrysarobin in others. Sometimes one of the two medications helps when the other fails. This makes anthralin quite indispensable in the practical treatment of psoriasis. It is, however, probably incorrect to say that it stains less or that it has a less irritating effect on the conjunctiva. Conjunctivitis is rarer only because anthralin is customarily prescribed in lower concentrations.

Some dermatologists maintain that chrysarobin and anthralin have better effects if one permits them to irritate (by using ointments instead of pastes or by the addition of salicylic acid). Obviously this is not true. Intentional irritations are unnecessary and not too rarely are dangerous. For about a generation *Dreuw's ointment* has been a great favorite in the treatment of resistant psoriasis (10 salicyl. acid 20 sapon. viridis, 20 chrysarobin 20 ol. rusci lanolin petrolatum āā). Dreuw's ointment is, however, weaker than an equally strong chrysarobin–zinc oxide ointment and has the disadvantages of heavier staining and more frequent irritations.

Most important in the treatment of chronic and resistant eczemas and psori-

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be powdered with alum, tannic acid resorcinol or well-localized judicious applications of resin of podophyllum (pure or 50 per cent zinc oxide) or better touched with 20 per cent resin of podophyllum in alcohol. The surrounding and opposite skin areas have to be carefully covered with a protective ointment or paste. Podophyllin is a very effective but also highly irritant drug which may cause severe contact dermatitis. Senile keratoses and xanthelasmata of the eyelids can be touched with liquefied trichloracetic acid after careful protection of the surrounding skin

Keratolytic agents —The removal of the horny layer frequently plays an important part in the therapy as, for example, in the treatment of old eczemas. It is most frequently accomplished with salicylic acid in the manner already described. But, for softening of calluses, corns, and other circumscribed keratoses, salicylic acid is used in salves, plasters, and varnishes. Alcoholic solutions and salves are used for peeling large surfaces, e g. in superficial mycoses, ichthyosis, keratosis palmaris. Other peeling agents are resorcinol (40 resorcinol paste) and beta-naphthol N F (10 beta-napthol, 20 sapon. virid., 50 sulf. precip. petrolatum) These pastes are used for systematic controlled peeling in acne sometimes in combination with other keratolytics. Soap especially the ointment like medicinal, soft soap (sapo mollis medicinalis U.S.P., green soap) has a very strong keratolytic quality However it is not so frequently employed as salicylic acid, mainly because it is difficult to dose properly

Disinfecting agents —Mercury iodine, and sulfur are common disinfectants used on diseased skin. Mercury is used as ammoniated mercury in ointments (10–33 per cent) or as mercury bichloride U.S.P in aqueous and alcoholic solutions and in salves (2 per cent and rarely more) and iodine as tincture of iodine (2 10 per cent i alcohol) The most important indications for mercury disinfection are the mycoses for iodine disinfection, mycoses and pyodermas. In pyodermas, especially in the follicular varieties sulfur has a much stronger effect though, in vitro it is inferior to the mercurials or iodine. I prefer to use it in combination with washings with alcohol as 33⅓ per cent sulfur in petrolatum in furunculosis especially to prevent recurrences. In streptococcal and staphylococcal infections of the skin ointments containing sulfonamides or penicillin take effect more rapidly but these ointments should be used only with much caution because of the danger of sensitization. Sulfonamide salves should not be used for longer periods than 5 days. Penicillin should not be used in ointments at all unless there is a special reason for it Systemic administration of sulfonamides and penicillin frequently fail to prevent recurrences (e.g in furunculosis) so that sulfur is quite indispensable for follow-up treatment. An old remedy which frequently does surprisingly well in the treatment of

1 Among less sensitizing, more modern, antibiotics effective in ointments against common bacterial skin infections are neosporin, bacitracin, polymyxin, tyrothricin, gramicidin, and the tetracyclines

atic residues is *salicylic acid* though its antieczematic effect is questionable. Strong salicylic acid ointments seem to exert a specific effect on some squamous eczemas. However the greatest importance of salicylic acid in external therapy is based on its keratolytic quality in high concentrations (5–20 per cent) Thus it is so to speak a pacemaker which guides the antieczematics to the deeper layers. This is all the more necessary because hyperkeratosis and parakeratosis are rarely absent in superficial chronic inflammations of the skin In the treatment of chronic eczemas salicylic acid is almost never used alone but usually in combination with the already named antieczematics In higher concentrations, it is incompatible with ammoniated mercury with which it forms the irritating sublimate The presence of zinc oxide interferes with the keratolytic effect of salicylic acid As an adjunct to antieczematics, it is therefore prescribed in greasy ointments, tinctures or varnishes.

OTHER MEDICATIONS

Besides the anti inflammatory drugs or antieczematics there is a large number of other chemically active substances which are often added to the described vehicles The most important ones will be briefly discussed

Astringents —Caustics in weak concentrations applied to eroded or ulcerated skin produce a fine and to the naked eye, invisible crust which covers and protects the denuded area Subsequently exudation is decreased and epithelization is enhanced Medicaments which have these effects are called *astringents* Agents of this kind most frequently used in dermatology are salicylic acid resorcinol tannic acid bismuth compounds potassium permanganate, aluminum acetate (in Burow s solution) and subacetate of lead (Goulard s extract) They are used for compresses in concentrations of 0 1–2 per cent and also in cooling salves. The most potent astringent is silver nitrate (0 0–0 1 per cent) an aqueous solution of which is used for wet dressing whereas erosions and fissures are cauterized with 2–10 per cent solutions. Silver nitrate solutions must be prescribed with distilled water since the natural sodium chloride content of many waters suffices to change the active silver nitrate into the relatively inactive silver chloride

Agents to stimulate granulation —Some of the same agents applied only in higher concentrations or in a more vigorous manner are used to stimulate the epithelization of ulcers. At the top of the list is silver nitrate in aqueous solutions (up to 10 per cent) for cauterizing or in the form of ointment dressings in paste or petrolatum (1–2 per cent) with balsam of Peru (10 per cent) This formula—1 silver nitrate 10 balsam of Peru in petrolatum—is known as *black* or *Billroth s ointment* Other pastes and salves which are said to stimulate granulation contain pellidol scarlet red and chlorophyll

Caustics —Silver nitrate in still stronger concentration or in stick form is used to cauterize granulations. Condylomata acuminata (venereal warts) can

U.S.P. (petroleum ether purified benzin—*highly inflammable!*) or by dusting with powder at night and brushing it out in the morning.

Agents against itching (antipruritics).—All vasoconstrictive agents have an effect on itching. Thus all cooling measures like open wet dressings, dabbing with alcohol, dusting with powder also reduce the sensation of itching. Well known home remedies are vinegar, rubbing with lemon slices, and household ammonia. Among important chemicals for relief of itching are menthol (½–2 per cent), thymol (½–2 per cent), and calmitol (10 per cent–pure). Phenol (1–2 per cent) is a strong antipruritic, and resorcinol (1–2 per cent) is also somewhat effective. All these medications are prescribed in alcoholic solutions and pastes. The experiences with antihistamines have not been uniform. Local anesthetics, especially novocaine and benzocaine (ethyl aminobenzoate U.S.P.), are also used, but they can be expected to be effective only on denuded skin and even there only temporarily. The external antipruritics often have to be supported by internal drugs such as antihistamines.

Bleaches.—Blackheads (comedones) can be bleached with hydrogen peroxide in solution or in salves. One may attempt to bleach hyperpigmentations by dabbing with 1–2 per cent mercury bichloride salve and to prevent their appearance by light-filtering substances, such as tannic acid (5 per cent in Aquaphor) or 5 per cent PABA (para-aminobenzoic acid) alcohol dilut.

Tinting agents.—Dyes are mainly used in *cosmetics* (hair dyes, lipsticks, rouge). In dermatology their role is more frequently that of a producer of skin diseases. As mentioned before, discolorations of the skin are frequently undesirable side effects in the application of dermatologic medicaments, especially chrysarobin, pyrogallic acid, tar, and sublimate. The only skin disease which, in the eyes of the practicing dermatologist, frequently needs artificial staining is vitiligo. A quite satisfactory way to darken the light areas is careful rubbing with 0.5 per cent potassium permanganate water. Of greater practical significance is the addition of red and brown chemically inert pigments to powders, lotions, and pastes to produce preparations of approximately normal color which may make it easier for the patient to stay on the job while he is being treated.

Anti-inflammatory agents.—Hydrocortone (hydrocortisone, 17-hydroxycorticosterone) is an adrenal glucocorticoid hormone which is highly effective in suppressing acute inflammations of the skin. Hydrocortone (¼–2½ per cent) is used in water-miscible ointments and lotions. It is suitable in well localized acute inflammatory dermatoses (e.g., eyelid and ear eczemas, pruritus ani). Its high price makes its use over large areas prohibitive, though some newer derivatives seem to be as effective in ¼ per cent strength as hydrocortone is in 2½ per cent concentrations. It is applied several times daily. Unfortunately, the effects of these anti inflammatory hormones are only suppressive, and rebound flareups are common when their use is discontinued.

pyodermas and the cleaning up of neglected ulcers is monoxide of lead N F (plumbi oxydum petrolatum āā) Other good disinfectants for infectious ulcerations are iodoform N F and strong phenol. All stronger antieczematics are also disinfectant agents especially the tars phenol (2 per cent) and chrysarobin Therefore they are suitable for the treatment of fungus diseases and other superficial infections. However *tars* should *not* be used in the presence of follicular pyodermas since they irritate the follicles (tar acne) and provoke recurrences In order to enhance disinfectant effects most of the named medications are combined with 5-20 per cent salicylic acid. Of course the same medicaments may also serve well for prophylatic purposes. I have already mentioned sulfur in this connection For the prevention of syphilis, mercury (sublimate washing and calomel salve) and quinine (30 quinine sulfate in Aquaphor) have been recommended but these applications are understandably unreliable

Skin irritants —Some agents are used to irritate the skin mainly in the hope that this procedure will stimulate hair growth This in part is the rationale for the use of tar preparations bichloride of mercury and tincture of capsicum in hair tonics Irritations of varying degree up to visible erythema and desquamation can be achieved in alopecia areata by painting with 2 per cent sublimate in diluted alcohol or tincture of iodine (2-10 per cent) Vigorous painting with tincture of iodine until a definite superficial dermatitis is obtained is sometimes done to stimulate the resorption of deep infiltrates Skin irritation with typical blister formation is accomplished by application of cantharidin in salves or plasters

Vasoconstrictive agents —Some chemical agents have the reputation of acting as vasoconstrictors e g ichthyol which therefore is used in pastes or ointments or alone in rosacea. However I have never seen any effect of this treatment on telangiectatic reddening Camphor (2-10 per cent camphor in alcohol or salve) monochlorbenzol (10 per cent in alcohol) and formalin (2-15 per cent in alcohol or salve) undoubtedly influence the blood vessels which explains their use in chilblains

Agents which decrease secretion —Formalin though it frequently irritates is used successfully to lower the *secretion of sweat* either in alcoholic solution (2-20 per cent) or as a dusting powder sometimes combined with tannic acid (Tannoform) Other chemicals serve the same purpose e.g aluminum chloride. The effect of chemicals on the *secretion of sebum* is much less reliable. Tannic acid and alum are recommended but I have not been able to convince myself of their effect. On the other hand washing with soap seems to stimulate the secretion of sebum which causes greasy hair After such washing the hair appears dry immediately afterward only to become greasy again faster than before. Therefore it is customary to have patients with greasy hair shampoo only once in 6 weeks and to degrease their hair in the meantime with petroleum benzin

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CHAPTER TEN

The Administration of the Treatment

It is nothing unusual in the external treatment of skin diseases that physicians simply prescribe some more or less complicated ointment formulas which they have memorized as students or have taken from a book and then wait to see what happens. There also is no doubt that this primitive method of treatment is frequently sufficient. Some eczemas disappear quickly when the patient, either on his own initiative or on a neighbor's suggestion, applies a little ichthyol paste, makes cold compresses, or lubricates the lesions with boric ointment. The same can be accomplished with prescriptions gleaned from a textbook of dermatology. Other eczemas, however, refuse to disappear and even become irritated or chronic, and then they often are extremely resistant to any therapy. These are the cases which come to the dermatologist and provide the justification for the special study and training in the field of external treatment of skin diseases. True, even in such cases one may occasionally have luck with prescribing an old fashioned and well tried recipe, e.g. Wilkinson's paste, but one may also make things much worse with it. The layman, of course, usually thinks that the doctor just "tries out" this or that prescription. This wrongs the dermatologist who has learned how to master his tools.

Of course, one may jokingly call *any* therapy "a trial," because certainty of success can never be guaranteed. But it makes a great deal of difference whether one uses a medication of more or less unknown value or one which experience has shown can improve or cure conditions of the kind in question. *Only in the* former case is one justified in speaking of a trial or experiment. In the latter case, however, we are dealing with a sensible therapy based on experience, i.e., trials in the past. This is valid, though even the best founded therapy never offers full certainty and, in addition, still carries the risk inherent in every potent drug, namely, that of causing toxic side effects. But it is a wrong conception held by laymen and some doctors as well that this factor of uncertainty is a special *characteristic of dermatotherapy. If one prescribes* a mere aspirin tablet, one never knows whether the patient will not become allergic to acetylsalicylic acid and develop the most alarming toxic symptoms, rare as this may be. And, however magically this drug may act in acute rheumatic arthritis, there are also failures which prompt the physician to change to other tried

medications. Nevertheless, it is true that in external treatment individual differences in reaction play a greater part than in the administration of medicaments by mouth and by injection. The dermatologist, therefore, must individualize much more. Also as the internist with all except bland medications starts with a small dose to satisfy himself that the drug is well tolerated in the particular case, so the dermatologist must first test the sensitivity of the skin before progressing to the application of stronger preparations. The art of the dermatotherapist lies in the *conscientious and methodical examination of the sick skin* and not in an intuition which tells him which salve is best. Therefore this "art" can be taught. Its principle is to start out by testing mild medicaments for their tolerance and therapeutic effect in a given case and then systematically to progress, on the basis of these tests, to stronger and finally to the strongest medicaments. Thus every therapeutic prescription should at the same time be utilized as a functional skin test. At each further step hypersensitivity or resistance may change the treatment plan. A conscientious dermatologic therapy therefore requires a daily, half weekly, or weekly checking of the patient.

Every prescription of medicaments should, of course, be preceded by a thorough *therapeutic history*. As far as possible it should be ascertained which therapeutic measures, which ointments and medicaments, have been used *so far* and what experiences have been had with them. In many cases it is a good idea to have the patient bring along the prescriptions of ointments used and then have him report about their individual effects. In this way one frequently gains knowledge of certain sensitivities, e.g., to petrolatum or mercury or sulfur, and also—what is almost more important—one learns which ointments and other medications have been well tolerated or even found agreeable without accomplishing a complete cure. These, then, are the suitable vehicles and medications with which one may begin planning treatment to force a cure by increasing the concentration, by intensification of the methods of application, or by combination with other medicaments.

Before doing all this, one should not forget to answer the first question of every therapy, namely, *whether to treat at all*. This question is an important one in dermatology because frequently one deals with conditions which cause little trouble and in which the treatment might be worse than the disease. If the patient exhibits a strong desire for treatment, the physician who declines treatment assumes the responsibility for driving the patient into the arms of quacks who would take advantage of him or even expose him to cosmetically dangerous procedures. In such cases the question arises whether the physician should stall and calm the patient with placebos, e.g., dabbing with salicylic acid alcohol or injections of turpentine. Justification of such things must be decided individually in each case. Certainly it is sometimes in the best interest of the patient if the physician follows the old maxim of *ut aliquid fieri videatur* ("let the patient see that something is being done"). However, in so doing it should be

completely clear that this rule contains considerable danger because it too easily begets the habit of routinely prescribing ineffective medicaments. In the end the physician may even fool himself into the belief that he has reached most wonderful results especially in diseases with naturally fluctuating courses, all the more so if other methods of treatment have been applied simultaneously Therefore I have always recommended not the renunciation of placebo therapy in selected cases but the clear indication of such use in the record so that it should not be forgotten that it *is only make believe therapy*

It is often necessary to use placebos temporarily in order to make good pre-treatment observation possible Some cases have been so changed by previous treatment that their true character can no longer be recognized A temporary placebo or make believe treatment with alcohol dabbings or bland compresses may therefore be necessary *for the diagnosis More frequently we have reason* to delay our treatment a little in order to form a clearer opinion about the *therapeutic* necessities Before starting to treat we really ought to try to find out what course the disease would take if *nothing* were done Failure to do this may cause serious errors. Thus for example some doctors fancy themselves to be in the possession of particularly good methods for treating boils, herpes pityriasis rosea Others treasure the effects of certain medicaments because they do not consider that the vehicle alone may have the same effect The necessity of not letting one s self be deceived is most urgent in the therapeutically more difficult hospitalized cases. Here fortunately a conscientious preliminary observation can most easily be carried out Therefore one should make it a rule not to treat hospitalized patients during the first few days if the spontaneous course of their *disease is not sufficiently known Or one may apply* treatment to only one side to compare the effect with the other untreated side In this way one is likely to find that some eruptions (e g atopic dermatitis, prurigo strophulus) heal surprisingly well in the hospital without treatment and that others (e g pemphigus) quiet down and improve surprisingly at *least for a while*

It is of great practical importance to establish that certain chronic eczemas (prurigo vulgaris of the French late exudative eczematoid of Rost *atopic* dermatitis of the Americans) may heal spontaneously if the patient is admitted to a hospital Just those cases which by their spontaneous tendency to heal in the hospital seem to be rather harmless have an unfavorable prognosis As a rule they recur a few days after discharge which upsets the patient s confidence in the physician *who has failed to predict this recurrence* The spontaneous healing on admission to the hospital and the tendency to recur on returning to the home conditions may be so pronounced that one may several times, *almost at will* make the patients heal in the hospital and relapse at home (Fig 346) The recurrences at home are presumably caused by allergens though so far it has not *been possible* to demonstrate these substances Bland treatments during hospitalization do not decrease the strong tendency to relapses. However such

relapses can often be prevented by more aggressive treatment, e.g., with tar (Fig 348) The tar treatment seems to make the skin more resistant. The chances for freedom from recurrence may be still further increased by proper continuation of treatment *at home* All this can be done systematically only if one has not overlooked the spontaneous healing in the hospital.

By carrying out preliminary observations with one-sided treatment, one is enabled to know the cases in which bland treatment does not accomplish a cure but prevents the eruption from *spreading* In these cases new eruptions may occur as soon as the treatment is discontinued or in other words, non-treatment or discontinuance of the treatment has a provocative effect.

Not only may a treatment be unnecessary as in the case of spontaneous healing but, though suitable it may be insufficient having only a suppressive effect on the progress of the disease but, beyond that, it may even be *harmful* by preventing spontaneous healing Such a *healing handicap* can be detected only if one side is at first left untreated If one treated the whole body one would not be able to recognize lack of improvement as a consequence of treatment. One would probably think that the treatment applied was *too weak* For example healing handicapped by external treatment would become apparent if an eczema healed on the blandly treated side, while persisting on the other side under mild tar and chrysarobin applications. In several mild cases of psoriasis which improved on zinc oxide paste only a similar zinc paste containing chrysarobin inactivated by soap prevented healing on the other side (Fig 349)

Thus it is not a matter of course to *treat* a patient, even in the beginning. In therapeutically difficult diseases, it is more important to wait and see first, at least for a while In this period of preliminary or pretreatment observation the dermatologist receives extraordinary help from leaving more extensive skin diseases unilaterally untreated or treating both sides differently This provides a control which *permits* one to judge exactly the success or failure of every method of treatment applied. Therefore *one side treatment* (Fig 350) is, in my opinion indispensable in all difficult cases which are treated externally Not only does this method provide a reliable answer to the question whether a treatment is necessary at all, but it also permits one to establish which medications are best tolerated and most effective in a particular patient. The one-side treatment (right-left treatment, simultaneous therapy) provides us with an

1 The expressions simultaneous therapy and "simultaneous paired comparison method" (Sulzberger) have also been suggested They are apt and expressive Frequently however especially in the beginning, actually only one side is treated. But, even if both sides are treated, one starts, in principle, with one medication, to which the other furnishes the control And, even if there is no exclusive one-side treatment, it does not matter so much that two skin areas are treated *at the same time* as that they are treated with *different medicaments*. Finally it should remain a leading viewpoint that not just any skin area but one side of the body is treated, with or without control treatment on the other side because comparing of non-symmetrical skin fields, i.e., in vertical direction, is not permissible except under exceptional circumstances For the reasons mentioned, the term "right left treatment also is not entirely correct, though it expresses the essential principle with certain humor

completely clear that this rule contains considerable danger because it too easily begets the habit of routinely prescribing ineffective medicaments. In the end the physician may even fool himself into the belief that he has reached most wonderful results especially in diseases with naturally fluctuating courses, all the more so if other methods of treatment have been applied simultaneously Therefore I have always recommended not the renunciation of placebo therapy in selected cases but the clear indication of such use in the record so that it should not be forgotten that it is only make-believe therapy

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C



D

Fig 315 *continued* — C Recurrence 5 days after discharge from hospital D spontaneous healing after second hospitalization



A



B

FIG 346.— *A* atopic dermatitis before hospitalization *B* spontaneous healing after hospitalization.

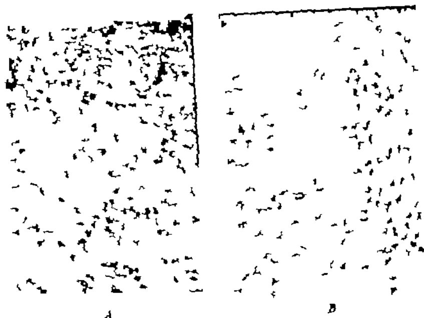


Fig. 348.—Immunization by treatment with tar. *A*, recurrence after bland lotion; *B*, no recurrence after coal tar.

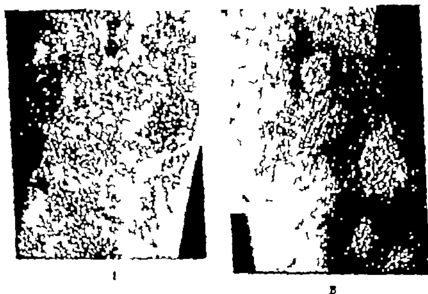


Fig. 349.—Healing prevented by soap-chrysarobin paste (*A*); healing on zinc oxide paste (*B*).

THERAPY BLANK FOR ONE-SIDE THERAPY

NAME JOHN DOE
DIAGNOSIS Atopic dermatitis
AGE 12

AFFECTED PARTS Face, elbows, trunk
TYPE OF LESIONS Papular erosive, lichenoid

1937	Arms R	Arms L	Trunk R	Trunk L	Legs R	Legs L	Face R	Face L	R	L	Systemic Therapy	Exam	Course Skin Disease
11/3	0	10 Zn vas	0	10 Zn vas	0	0	10 Zn vas.						
11/4													
11/5											Seconal at light gr 1½	Blood	
11/6													
11/7	10 Zn vas.	Liq carb	10 Zn vas	< 2 Tar Zn vas.									
11/8													
11/9													
11/10													
11/11				< 25									
11/12	=		33 Sulf vas.										
11/13													
11/14													
11/15													
11/16													
Right side no better													Some squamous remnants only

< means left side more improved than right side. > means right side better than left. 0 = no medication. = means equal on both sides.

FIG. 347

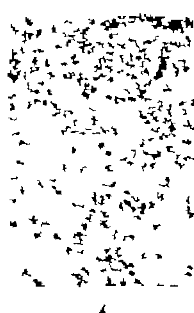




Fig. 348.—Immunization by treatment with tar: *A*, recurrence after bland lotion; *B*, no recurrence after coal tar.





Fig. 349.—Healing prevented by soap-chrysarobin paste (*A*); healing on zinc oxide paste (*B*).

individual *reactional therapy* Failing to make use of it in difficult cases may lead to errors about the effect of the medications used

Of course, one should not think that by the use of *the one-side treatment*, one can simply read from the control side what one needs to know The correct execution of the method and the appraisal of its results are not easy The method furthermore is not very suitable for ambulatory treatment *It took me* 6 years of fruitless experiments before I finally acquired the "know how" of the

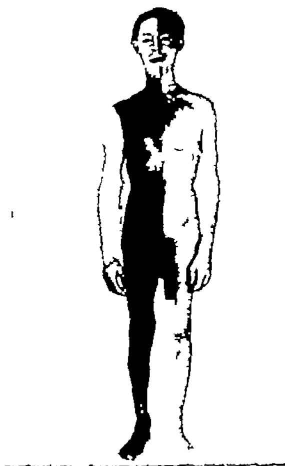


FIG 350 —One side treatment (8 fields method)

method This difficulty is the reason why the occasional observations on one-side treatment by earlier dermatologists have not led to useful results *This method like anything else requires learning* One must use it regularly and systematically to become an expert The evaluation of the observations obtained must be done carefully and critically

The one side treatment may be executed in the form of right left experiments as six fields or eight fields or as cross-experiments but one should always beware of outlining *too small fields* A comparison of non-symmetric fields is not permissible because of the inherent increase in sources of error One should avoid

smearing into adjoining fields and, if irritations occur one should be alert for traveling, jumping to symmetric areas, and distant irritations. During the right left treatment of the legs, the patient must wear pajama trousers to prevent transfer from one leg to the other. Above all all important observations must, within reasonable limits, be confirmed by *repetitions and/or reversal of the sites of the experiment*

One always has to make sure that the right-left applications do not differ in *more than one ingredient* Thus one should not treat with zinc oxide oil on one side and tar zinc oxide paste on the other side but should always pair e.g., zinc oxide oil and tar zinc oxide oil or zinc oxide paste and tar zinc oxide paste. Otherwise one runs into an "equation with two unknowns, which makes the evaluation much more difficult, if not impossible. In order to get distinct results one should treat with *maximal strength* on the side to be tested for the more active ingredient or in some cases, on both sides with maximal strength. Insignificant differences between the two sides should be disregarded and not recorded because the recording of uncertain results causes confusion.

The time for the execution of the experiment should not be too short. One should allow at least 2 weeks and follow this up by a treatment-free *observation period* An orderly one-side treatment is impossible without the use of special *therapy blanks* with individual columns for the treatment of arms, trunk, legs face etc each separate for right and left (Fig. 350) The differences between the sides are suitably entered with symbols such as = or > or < Other findings are entered on the other side of the sheet or dictated to a stenographer during the rounds. Valid examinations with this method are impossible without the benefit of trained interested and reliable nurses. If one wants to draw general conclusions from the observations they must be *statistically analyzed* With all these precautions, the treatment of skin diseases certainly can be made more effective than with the uncontrolled and therefore, really blind conventional manner

After the preliminary or pretreatment observation or in the case of one-side treatment simultaneously the treatment with vehicles alone is started, which is entirely with physical and mechanical agents. I call this the *bland starter treatment* The purpose of this phase is to establish which vehicle is best tolerated and most effective in the particular case. Of course there exist certain general rules for the choice of vehicles as has already been pointed out. For example in acute and especially in weeping dermatitis salves and powders frequently irritate while lotions and zinc oxide oil do not (Fig 351) However a case may occasionally react in the opposite way and tolerate zinc oxide petrolatum better than lotion (Fig. 352) This can easily be found out by methodical one-side treatment. If it has once been established which type of vehicle is the best for the case to be treated and the treatment with it proves to be insufficient then one progresses to the proper therapy by adding *chemically active* medica

1. For explanation of symbols see p. 242





Fig. 351.—Acute eczema typical reaction. Zinc oxide petrolatum irritates (*A*) while shake lotion heals (*B*)





Fig. 352.—Atypical reaction in acute eczema zinc oxide petrolatum (*A*) is better tolerated than shake lotion (*B*)

ments to the vehicle which was found most satisfactory Before doing so it is suitable to do patch tests with the active medicaments which one plans to use. By this method one may be able to establish specific hypersensitivities, e.g to mercury which may save the patient later irritations. Thus the preliminary observation and starter treatment periods are followed by *medication patch testing*

The medication patch testing is done by attaching to the skin with adhesive tape lentil-sized (0.5 cm.) patches which have been impregnated with the re-

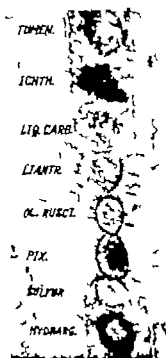


Fig 353 —Standard series of patch tests Tumenol is bituminous tar Liantral is 10 per cent solution of coal tar in benzol

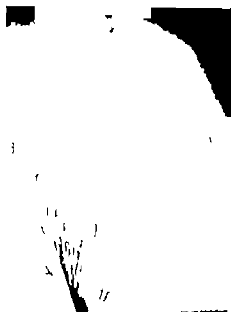


Fig 354 —Series of patch tests in hypersensitivity to coal tar

spective medicaments. These patches remain in contact with the skin for 24 hours. The result is read 48 hours later I use a standard series of medicaments consisting of different tars, resorcinol, sulfur and mercury (Fig 353) The sensitivities discovered in this way may be very unexpected. For example I had a patient with psoriasis who tolerated without trouble the strongest medications, such as 33 per cent mercury 20 per cent chrysarobin, 10 per cent pyrogallic acid or 10 per cent wood tar but reacted to coal tar preparations (liquor carbonis detergens and pure coal tar) with violent inflammations (Fig 354) Of course to save time the patch testing can be done during the bland starter treatment Finally now the point has been reached at which an active chemo-



A



B

Fig 351 — Acute eczema typical reaction. Zinc oxide petrolatum irritates (*A*) while shake lotion heals (*B*)



A



B

Fig 352.—Atypical reaction in acute eczema, zinc oxide petrolatum (*A*) is better tolerated than shake lotion (*B*)

pyrogallic acid can be so firmly fixed to the skin that they stain the linen but little. Varnishes and tinctures may be covered with adhesive tape, which at once eliminates all such inconvenience. This method can however be used only in small solitary foci. With the use of pyrogallic acid and chrysarobin, staining may create real problems. Of course, such ointments cannot be applied *with the fingers* because they would stain the hands and nails black. It is practical to use toothbrushes or tongue blades for their application. If an ambulant treatment is still deemed feasible one should start by telling the patient to wear his oldest underwear during the course of the treatment. Underwear soiled with chrysarobin or pyrogallic acid must be washed separately to prevent staining the other family laundry. Yet, for a stronger treatment of larger areas with chrysarobin these precautions are insufficient because the chrysarobin penetrates everywhere, staining beds, furniture and articles of daily use. If more extensive applications of chrysarobin are planned, the *staining alone* is a strict indication for hospitalization. Of course, the hospital must be well prepared for this type of treatment. The patients not only must receive their own, usually brown underwear and bed clothes, which have to be laundered separately but also clothing which can be tied with ribbons at the wrists and ankles. At night, the patients must wear gloves and goggles for the protection of the conjunctivae. Thus a special "chrysarobin dress" is necessary. Therefore, stronger treatments of psoriasis can generally be carried out properly only in dermatologic hospitals or wards.

It is a peculiarity of some preparations that they appear colorless but still stain after they have been applied, even days or weeks later. This is well known of silver nitrate, but it is also of practical significance in treatment with mercury. At first, it seems rather innocuous to treat the hair with strong ammoniated mercury ointments or sublimate alcohol. However under the influence of light, a very ugly slate discoloration of the fingernails appears days or weeks later which cannot be removed. Therefore the patient should from the start use finger cots when applying the medication. Some discolorations appear only under certain conditions. It is necessary to know that hair treated with sublimate alcohol may after a permanent wave, by the formation of mercury sulfide suddenly turn greenish black. To prevent such embarrassing surprises it is good practice to alert blond or white-haired patients who are under treatment with mercury preparations. The applicability of some preparations is decisively influenced by their *odor*. Birch tar (oleum rusci) *the classical tar preparation* for the treatment of chronic eczemas, has such a strong odor that it cannot be used ambulatorily in more extensive skin diseases. Coal tar does not have this disadvantage. Some preparations, like sulfur smell only when they get warm e.g. if applied to intertriginous areas. *The* odor of others—for instance phenol—is so volatile on the body as well as on the hands, that it is only slightly embarrassing. In hair however it lingers on for a very long time

therapy can be started according to the information already obtained Since we always have at our disposal a great variety of *preparations the choice of the preparation* needs some careful consideration Of course there are two properties of the medicament which play a decisive part in this selection namely its therapeutic power and its toxicity including its irritating effect This is a general rule in medicine In dermatology however we frequently must consider qualities which have *nothing to do* with the main purpose of healing the disease I refer to *staining* and *odor* In certain cases it is simply impossible to use some otherwise excellent drugs because of the *staining* which they cause. This of course, refers particularly to the ambulatory *and not the hospital treatment* of exposed parts especially the face and hands. Therefore the dermatologist must be well acquainted with the staining power of the medicaments which he prescribes Some preparations which are otherwise closely related may cause very different discolorations e.g pyrogallic acid and lenigallol while other related preparations such as chrysarobin and cignolin stain about equally strongly though most textbooks claim that there is a great difference with less staining from cignolin Some drugs bring about a *peeling* either alone or together with the discoloration (e g resorcinol) *which interferes with their application* just as much

Of course discoloration and peeling also depend on the concentration of the drug and on the *vehicle* in which it is prescribed For instance pure ichthyol is pitch black If it is used in a 10 per cent ointment as is still frequently done *in the treatment of pyodermas everything gets soiled However if it is used in* paste or lotion then it stains only a moderately dark brown If one prescribes only 2 or 3 per cent with as little as 10 per cent zinc oxide one obtains an almost skin-colored light tan ointment An addition of ichthyol to zinc oxide salves and lotions in this quantity therefore facilitates the application of these vehicles to the face since the brilliant white color of lotion and zinc oxide oil compels the ambulatory patient to interrupt his treatment by frequent cleansing of his face On the other hand the addition of zinc oxide in small quantities *causes pyrogallic acid even in only 1 per cent ointment* to stain everything black Combination with other medications may influence the staining power as much as the vehicle Thus it is claimed that acid (citric acid) reduces the staining power of pyrogallic acid—a claim which I have been unable to confirm Chrysarobin becomes a dirty black on the addition of soap (alkali) The effects of the combination *of different drugs with regard to discoloration* must be sufficiently known to the prescribing doctor or he may be in for disagreeable surprises On covered parts of the body the staining of the skin is, of course of no great importance But it is important to what degree the preparation which has been applied to the skin is absorbed by the garments This also depends partially on the vehicle. Discoloring *drugs in greasy ointments stain* the underwear more than in pastes. By the use of collodion some staining medicaments e.g



Fig. 155.—Irritation from chrysarobin, erythematous



Fig. 156.—Irritation from pyrogallic acid, pustular

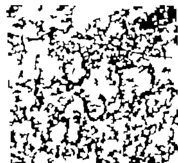


Fig. 157.—Distant irritation, eczematoid type (from sulfur)



Fig. 158.—Chronic folliculitis from coal tar (so-called tar acne)



Fig. 159.—Granular keratosis from coal tar on upper arm

making the addition of 2 per cent phenol to a hair tonic impossible. The m important considerations in the choice of a preparation are, of course *lancu* and *healing* effect As for the irritative effect each preparation has its own rule This is the *normal irritative effect* Its frequency and strength generally but no always run parallel with the intensity of the healing effect, which means th preparations with vigorous antieczematous and antipsoriatic effect are ab more liable to irritate Therefore we encounter irritations most frequently i using the *strongest preparations on our scale (mercury tar pyrogallic acid* chrysarobin) Again we find that closely related preparations may behave very differently Bichloride of mercury (sublimate) irritates quite regularly in concentrations of more than 2 per cent while mercurous chloride (calomel) almost never irritates. Ten per cent pyrogallic acid elicits irritation in the majority of cases while 20 per cent lenigallol does so extremely rarely Not only the frequency but also the character of the irritation is characteristic of the medica ment We have already mentioned the explosive erythematous type of irritation from chrysarobin (Fig 355) the late (appearing only after 8–10 days) pustular irritation from pyrogallic acid (Fig 356) the tendency of sulfur to produce erythemato-squamous distant irritations of *craquelé* character which *peculiarly often spare the actually treated areas (Fig 357) The tars especial* ly coal tar if used over long periods of time frequently make chronic papular folliculitis so-called tar acne which is asymptomatic but recedes only very slowly (Fig 358) Other sequels of tar treatment include comedones, follicular pustules follicular and also diffuse ichthyosiform squamous or verrucous keratoses (Fig 359) Naturally the sensitivity of the skin differs in various regions. In this respect too the individual medicaments display special fea tures. Irritations from pyrogallic acid and chrysarobin frequently affect the flexures of the large joints. Coal tar often irritates when applied to the face. The dangerous consequences of getting chrysarobin into the conjunctival sac have already been mentioned Thus there exists a *topography of irritability* knowledge of which is frequently of practical significance. The toxic effects of the medicaments applied to the skin may of course extend to *other organs* Ointments containing salicylic acid mercury and tar may cause irritations of the kidneys with albuminuria and cylindruria if they are used on large sur faces The urine may turn green black by oxidation of free phenols (phenol urine) If because of lack of supervision and urine examination treatment is continued beyond this stage severe poisoning with nephrosis and coma may ensue However contrary to the claims of many textbooks chrysarobin is not dangerous to the kidneys Mercury ointment may even without skin irritation cause a violent stomatitis such as is well known as a sequel to mercury injec tions and inunctions with blue ointment in the treatment of syphilis. Pyrogallic acid not only causes renal irritation but also is a blood poison This toxic effect however occurs only after the caustic treatment of denuded surfaces (lupus



Fig. 355 Irritation from chrysarobin, erythematous



Fig. 356.—Irritation from pyrogallic acid, pustular



Fig. 357 —Distant irritation, *craquelé* type (from sulfur)

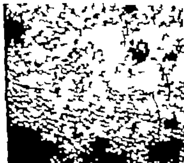


Fig. 358.—Chronic folliculitis from coal tar (so-called tar acne)



Fig. 359 —Granular keratosis from coal tar on upper arm

making the addition of 2 per cent phenol to a hair tonic impossible. The most important considerations in the choice of a preparation are of course, *tanicity* and *healing effect*. As for the *irritative effect*, each preparation has its own rule. This is the *normal irritative effect* Its frequency and strength generally, but not always run parallel with the intensity of the healing effect, which means that preparations with vigorous antieczematous and *antipsoriatic effect are also* more liable to irritate. Therefore we encounter irritations most *frequently in* using the *strongest* preparations on our scale (mercury tar pyrogallic acid *chrysarobin*) Again we find that closely related preparations may behave very *differently* Bichloride of mercury (sublimate) irritates quite regularly in *concentrations of more than 2 per cent* while mercurous chloride (calomel) almost never irritates. Ten per cent pyrogallic acid elicits irritation in the majority of cases while 20 per cent lenigallol does so extremely rarely Not only the frequency but also the *character of the irritation is characteristic of the medicament* We have already mentioned the explosive erythematous type of irritation from chrysarobin (Fig 355) the late (appearing only after 8–10 days) pustular irritation from pyrogallic acid (Fig 356) the tendency of sulfur to *produce* erythemato-squamous distant irritations of *craquelé* character which *peculiarly often* spare the actually treated areas (Fig 357) The tars, especially coal tar if used over long periods of time frequently make chronic papular folliculitis so-called tar acne which is asymptomatic but recedes only very slowly (Fig 358) Other sequels of tar treatment include comedones follicular pustules follicular and also diffuse ichthyosiform squamous or verrucous keratoses (Fig 359) Naturally the sensitivity of the skin differs in various regions In this respect too the individual medicaments display special features. Irritations from pyrogallic acid and chrysarobin frequently affect the *flexures of the large joints* Coal tar *often irritates when applied to the face.* The dangerous consequences of getting chrysarobin into the conjunctival sac have already been mentioned Thus there exists a *topography of irritability* knowledge of which is frequently of practical significance. The toxic effects of the medicaments applied to the skin may of course extend to *other organs* Ointments containing salicylic acid mercury and tar may *cause irritations* of the kidneys with albuminuria and cylindruria if they are used on large surfaces The urine may turn green black by oxidation of free phenols (phenol urine) If because of lack of supervision and urine examination treatment is continued beyond this stage severe poisoning with nephrosis and coma may ensue. However contrary to the claims of many textbooks, chrysarobin *is not* dangerous to the kidneys. Mercury ointment may even without skin irritation cause a violent stomatitis such as is well known as a sequel to mercury injections and inunctions with blue ointment in the treatment of syphilis. Pyrogallic acid not only causes renal irritation but also is a blood poison This toxic effect however occurs only after the caustic treatment of denuded *surfaces* (lupus

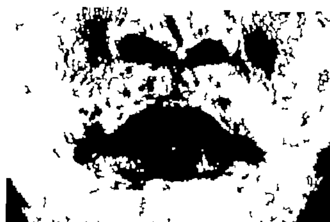
sensitivity may be directed against a variety of drugs (polyvalent) or it may be strictly specific (monovalent) In the latter case, only one of a group of chemically closely related agents may irritate while the others fail to do so For instance coal tar but not wood tar chrysarobin and not cignolin may irritate or vice versa. By the use of routine patch tests for drugs, pronounced sensitivities of this kind can be detected before they cause trouble. Difficult problems may arise if sensitivities which were absent when the treatment was started develop during the course and from *the treatment* In this case we speak of *acquired sensitivity* or *sensitization*. Some preparations are such powerful sensitizers that, in general they cannot be used in ointment form or at the most, only for a few days in succession. This is true of the sulfonamides, antihistamines, local anesthetics, and penicillin But even the most commonly used antieczematics may sensitize. I saw a patient whose extensive eczema healed on tumenol (a tar resembling ichthyol) except for a small residue on one calf Just when the healing had progressed that far he developed a hypersensitivity to tumenol, starting with a tumenol irritation on the leg but soon followed by dissemination over the entire body Afterward other tars irritated, too so that only sulfur could be used to overcome the dermatitis. Of course one may observe the same thing in the course of less irritable dermatoses. For instance I treated an acne patient for months successfully with 20 per cent ammoniated mercury Suddenly he became so sensitive to mercury that 1 per cent ammoniated mercury lotion caused the most violent inflammation. Such cases show that finding a single drug sensitivity does not free the dermatologist from the obligation to remain alert for new allergic developments. Throughout the entire duration of the treatment, he must observe untiringly and carefully and if necessary test again and again.

Under treatment entirely local hypersensitivities may develop for instance by starting with too strong and irritant concentrations. This is illustrated by the following example In a boy who was treated with 2 per cent Liantral (10 per cent coal tar benzol) paste an irritation occurred on the left side of the forehead only where 20 per cent Liantral lotion and pure Liantral had previously twice caused irritations, while the right side which so far had been treated with milder medications, remained quiet (Fig 361)

All the sensitizations discussed so far were directed against the same agent which elicited them (homotypic) However there also are *heterotypic sensitizations* For instance after treatment with eosin alcohol, I observed that a patient had become sensitive to Pellidol, to which he had not been sensitive before. Even chrysarobin which is the prototype of a *desensitizing* medicament (see below) is able to elicit hypersensitivity I had a patient with psoriasis who at first had had a mild irritation from 2 per cent chrysarobin paste but later tolerated 20 per cent chrysarobin paste 25 per cent Liantral, 10 per cent pyrogallic acid, and 33 per cent ammoniated mercury without trouble. But when

vulgaris) and not after application to the intact skin which probably does not permit absorption of significant amounts

The irritations which have been discussed so far are so to speak qualities of the respective medicaments There is however a type of irritation in whose genesis not so much the medicament as a specific sensitivity of the patient appears to be the most important causal factor Such atypical frequently



A



B

Fig 360 —*A* hypersensitivity to boric acid boric acid in petrolatum on right side of patient's mouth irritates, while petrolatum alone on left side does not. *B* the same reaction was elicited with the sides reversed Boric acid in petrolatum on left side, petrolatum alone on right side

highly specific hypersensitivities may be caused by the most innocuous and mild medicaments even by boric acid which in general is tolerated by the most irritable eczemas (Fig 360) Certain drugs more frequently cause such specific sensitizations so that one must always be alert to them as for example in the case of mercury Some sensitivities are encountered more often in one country than in another I observed sensitivity to sulfur much more often in Holland (Leiden) than in southern Germany (Munich) The hyper

sensitivity may be directed against a variety of drugs (polyvalent) or it may be strictly specific (monovalent). In the latter case only one of a group of chemically closely related agents may irritate, while the others fail to do so. For instance coal tar but not wood tar, chrysarobin and not cignolin may irritate or vice versa. By the use of routine patch tests for drugs, pronounced sensitivities of this kind can be detected before they cause trouble. Difficult problems may arise if sensitivities which were absent when the treatment was started develop during the course and from *the treatment*. In this case we speak of *acquired* sensitivity or *sensitization*. Some preparations are such powerful sensitizers that, in general, they cannot be used in ointment form or at the most only for a few days in succession. This is true of the sulfonamides, antihistamines, local anesthetics, and penicillin. But even the most commonly used antieczematics may sensitize. I saw a patient whose extensive eczema healed on tumenol (a tar resembling ichthyol) except for a small residue on one calf. Just when the healing had progressed that far he developed a hypersensitivity to tumenol, starting with a tumenol irritation on the leg but soon followed by dissemination over the entire body. Afterward other tars irritated too, so that only sulfur could be used to overcome the dermatitis. Of course one may observe the same thing in the course of less irritable dermatoses. For instance I treated an acne patient for months successfully with 20 per cent ammoniated mercury. Suddenly he became so sensitive to mercury that 1 per cent ammoniated mercury lotion caused the most violent inflammation. Such cases show that finding a single drug sensitivity does not free the dermatologist from the obligation to remain alert for new allergic developments. Throughout the entire duration of the treatment, he must observe untiringly and carefully and, if necessary, test again and again.

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he suffered an irritation from sublimate he reacted to 0.2 per cent chrysarobin paste with an oozing dermatitis and even 0 05 per cent caused a painful erythema Of practical importance is sensitization *to light which is frequently caused* by certain chemicals e g eosin coal tar sulfonamides Therefore the face should not be treated with these agents particularly not in summer and in the tropics. Sensitization may be frequent with *one vehicle and almost non-existent* with another as in the case of sulfathiazole the administration of which in ointment form is dangerous while alcoholic solutions are so innocuous that it is used as a sunburn protective for sun bathers. Of *course related agents may again behave* quite differently Coal tar solution in benzol photosensitizes regularly while an extract of the same tar in tincture of quillaja (liquor carbonis detergens) fails



Fig 361 —Local sensitivity to coal tar on the forehead

to do so Such differences may determine the suitable choice of a method of treatment

During the course of treatment not only a sensitization but also a *desensitization* may take place. This is very regularly the case with many remedies e g chrysarobin so that we consider this getting used to it right from the start whenever we treat with chrysarobin Therefore we start with weak concentrations and progress to stronger ones only after the weaker ones have been used for a certain time Because of the tolerance by *habituation* we *should not* stay too long on weak concentrations. A chrysarobin paste of a certain strength to which the skin has become accustomed usually no longer exerts a healing effect The development of tolerance to the remedy *therefore is the* rationale for treatment courses with gradually and slowly increasing strengths of chrysarobin which are customary in the therapy of psoriasis

Acquired tolerance to a certain *medicament may be accompanied* by an acquired tolerance to other drugs this being also heterotypic (polyvalent) For example I could make a cignolin intolerant patient tolerant to this agent by the application of concentrated *chrysarobin pastes*.

Intolerance to medicaments does not necessarily manifest itself only in irritations. It may also cause *provocations* the irritated areas changing into characteristic lesions of the respective disease. This, for instance is the case when new psoriatic lesions develop in the site of positive pyrogallic acid tests (Fig 362) This is "provocation by irritation" which we have already discussed.

An extension of the affected areas may also be provoked by treatment without demonstrable irritation. I have called this phenomenon *silent provocation* Of course it can be ascertained only by one-side treatment. For instance we saw psoriasis progress on the side treated with 0.5 per cent chrysarobin paste while the other side, which was treated with paste only remained stationary



Fig 362.—Provocation by irritation (Koebner phenomenon) in psoriasis. Positive patch tests with pyrogallic acid turn into psoriasis.

(Fig 363) There was neither chrysarobin irritation nor chrysarobin sensitivity All these facts show that we are never safe if we unhesitatingly apply the medicament which seems to offer the greatest chance of cure. As in other branches of medicine the *dangers of toxicity and the therapeutic chances* must be weighed against each other If the danger of poisoning is considerable one will prefer a less good, but more innocuous, remedy If staining power odor and toxicity do not influence our choice of a remedy we will, *of* course, select the preparation which offers the best chances for cure in the disease to be treated. The *average expectancy* of our medicaments in the individual diseases is frequently well known For instance resorcinol and sulfur are outstanding remedies for many eczemas but have little or no influence on psoriasis. Thus one must not only progress from mild to stronger agents but also select as far as possible, the

most promising one for each disease and stage If against our expectations, the case proves to be refractory then we have to fall back on the less-well-proved drugs. This implies that in the course of a longer treatment one is likely to slide gradually from the preparations with the greatest healing chances to those with smaller and little chances For example in most eczemas one will start active treatment with tar which is the most reliable antieczematic. If the case proves hypersensitive or refractory one will use phenol resorcinol sulfur or mercury in the order named because these are the drugs which very frequently have an antieczematic effect though not so regularly as tar In very chronic cases the antipsoriatics may occasionally be tried first. If the case still proves to be sensitive or refractory we will for example prescribe tannic acid i.e. a drug which though not an antieczematic proper is occasionally effective in eczema

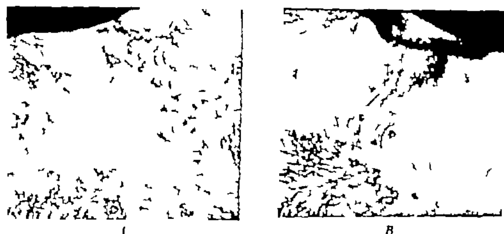


Fig 363 — Ilent provocation by ½ per cent chrysarobin paste on right side (I) zinc paste only on left side (B)

If this also fails we will resort to medications whose antieczematic effect is not established at all but which have a vague reputation that here and there a case of eczema responded well like Pellidol or iodine In the interest of the patient one should by all means avoid the reverse direction of haphazardly trying first medications of medium or little probability of success (e.g mercury or tannic acid) before progressing to the relatively more reliable ones. The situation becomes more complicated by the fact that various skin diseases respond quite differently according to their sites It is just as correct to speak of a *topography of healing effect* as it is to speak of a topography of irritation. With ammoniated mercury and liquor carbonis detergens, for instance, one can frequently heal psoriasis of the face while lesions on the elbows and knees only rarely respond to these medications

In order to carry out an orderly treatment it is therefore necessary to know the degree of healing probability of the selected medicament in the particular disease compared with other medications. Chronic papular eczemas for in

stance, are more frequently and rapidly healed by coal tar than by wood tar psoriasis can quite regularly be influenced by pyrogallic acid and only exceptionally by lenigallol (Fig 365) Yet the probable course of the treatment is still not clearly enough indicated in a given case by such statistical comparative healing effects of our medications. Besides these ***normal therapeutic responses***

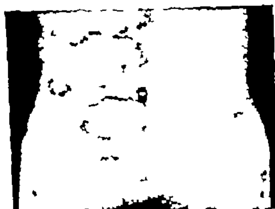


Fig. 364 —Normal response Psoriasis healed by pyrogallic acid (*left side of patient*) and not influenced by Lenigallol (*right side of patient*)



Fig 365 —Paradoxic response Psoriasis healed by Lenigallol (*left side of patient*) while right side which had been treated with pyrogallic acid, still squamous

(Fig 364) there exist in some cases, atypical or ***specific therapeutic responses*** which must be discovered by systematic investigations. Following is an example of this phenomenon The best remedy against psoriasis is chrysarobin Yet I know of a case in which chrysarobin as well as pyrogallic acid, mercury and birch tar was ineffective while, over an observation period of two decades, each recurrence has quickly been controlled by coal tar In another case in which all

customary antipsoriatics and also X ray treatments were of no avail many small recurrences on the lower legs could be promptly healed with pyrogallic acid This was ascertained time and again over twenty-five years. One-side treatment is the indispensable method for the discovery of such specific therapeutic responses.

The specific therapeutic response may of course also *be limited to a certain region* Psoriasis may respond to chrysarobin paste faster on the trunk than on the thighs or an eczema whose lesions look alike everywhere may tolerate zinc oxide petrolatum on the trunk better than lotion while on the extremities lotion is better tolerated than zinc oxide petrolatum Once I observed that a psoriasis of the thigh healed quicker under dressings with chrysarobin paste than on open application of the same paste while the lower leg of the same patient showed the opposite response. It may happen that most of the lesions in a case of psoriasis heal without difficulty with only mild treatment with chrysarobin while in one area a group of lesions proves resistant and yields only to the strongest combination treatment. Thus the *topography of therapeutic response* may also exhibit marked individual peculiarities. Just as hypersensitivities may come and go so the therapeutic response is *not an unchangeable quality* of the skin though I do not know of an example of a development of a therapeutic response which did not exist before. However the *vanishing of a therapeutic response* which was originally present may be observed frequently Acne, for instance may heal almost completely on resorcinol but relapse under continued treatment. Psoriasis, at the end of a successful treatment with chrysarobin may become chrysarobin resistant In such cases we say that the eruption has become *refractory* or *resistant* This of course compels us to change the preparation hoping that the resistance will disappear after a long period of treatment with other remedies and that the therapeutic response to the original drug will be restored Unfortunately we do not know how well founded this hope is

In ailments with a tendency to recur (psoriasis eczemas) not only do we try to make the present eruption vanish but we also want to change the tune of the skin so that new eruptions no longer occur We desired to accomplish a permanent effect an *immunization* The healing effect and the immunizing effect of an agent do not necessarily run parallel. Some eczemas heal on bland treatment just as fast as on tar medication However after bland treatment the eruptions recur which is not the case after tar treatment (Fig 348) One may observe a corresponding phenomenon if one treats one side with wood tar the other with coal tar (Fig 366) It is of considerable importance that the addition of certain agents to an ointment or the preceding treatment with them may though not interfering with the healing prevent the immunization We saw such a prevention of immunization in a psoriatic who after unsuccessful treatment with sulfur petrolatum and sulfur-soap-petrolatum quickly healed

on cignolin paste, but shortly thereafter developed an extensive recurrence on the side which had been treated with soap while the other side remained free (Fig 367) The differences with regard to the immunizing effect must therefore also influence our choice of the drugs. It must, however be admitted that our knowledge in this field needs further corroboration and extension by systematic one-side treatment.

All these considerations have a bearing on the choice of a preparation. But we must, in each case also determine the optimal *concentration* The dose as in all other fields of medicine is of the greatest importance. If we are too cautious, we waste time with ineffective treatment. If we treat too vigorously we risk poisoning which in our case, means irritation acquired sensitivity and provocation In determining the concentration we are confronted with the same questions as those we faced at the selection of the drug Discoloration and odor depend to some degree on the concentration of the remedy but more important is the influence of the concentration on irritating and healing effects.



Fig 366—Intertriginous... Recurrence after wood tar (*A*) no recurrence after coal tar (*B*)

The *irritating effect* of course increases with the increasing concentration. This is the reason why active treatment of eczemas is started with low concentrations, which are then progressively increased when the lower strength has been tolerated or the skin has become used to it.

The *healing effect* also usually increases with the concentration. Therefore we systematically try to increase the concentration as much as possible in all

chronic treatments. However we dermatologists have to consider the fact that some of our medicaments in higher concentration not only exert a stronger but frequently an entirely different effect than in low concentration We have already mentioned that many astringents become caustics at higher concentrations (silver nitrate resorcinol) i e. the same medicament in a weak dose stimulates the formation of epithelium but in a strong dose destroys it. Salicylic acid acts similarly Under 2 per cent it promotes keratin formation (keratoplastic) Furthermore the effect of a given medicament at the same concentration may be quite different on unbroken skin and on eroded or ulcer

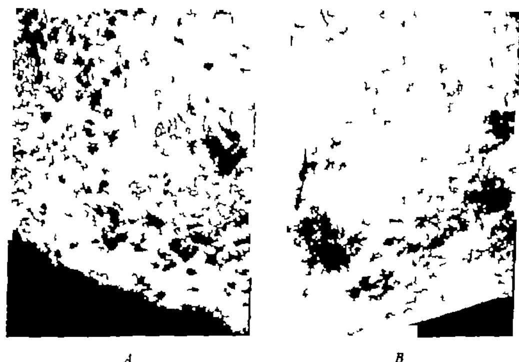


Fig 367 —Prevention of chrysarobin immunization by preceding treatment with soap recurrence on the soap-treated side only (A)

ated surfaces For example this is true of pyrogallic acid which as a 10 per cent ointment on intact skin acts as an antieczematic and antipsoriatic but becomes a strong caustic on eroded skin Therefore one must be on guard not to continue the treatment of psoriasis with pyrogallic acid if fissures develop The *choice of the vehicle* also significantly influences the effect of the medication with regard to staining irritation and healing effect It has already been mentioned that pyrogallic acid in collodion soils less than in ointments. Chrysarobin in collodion irritates much more than in pastes but nevertheless exerts a weaker healing effect The type of ointment base used in pastes and salves may alone be of importance for the healing effect. For example chrysarobin and cignolin almost never irritate in lanolin and lanolin pastes but quite regularly do so in

petrolatum and petrolatum pastes. This difference is so great that it can be demonstrated in tests (Fig. 368). For example, chrysarobin and cignolin (anthralin) have a stronger effect on psoriasis if applied in petrolatum than in lanolin (Fig. 369). Such differences must be taken into consideration in order to obtain the optimal effect. By the way the systemic *absorption* of many medicaments and therefore systemic toxic effects also depend on the ointment bases used. For instance salicylic acid is absorbed much more from Aquaphor than from lard, but without at all enhancing its dermatotherapeutic e.g. keratolytic action. On the contrary it has been found that the extent of ab-



Fig. 368.—Irritation from cignolin in petrolatum (A). failure to irritate in lanolin (B).

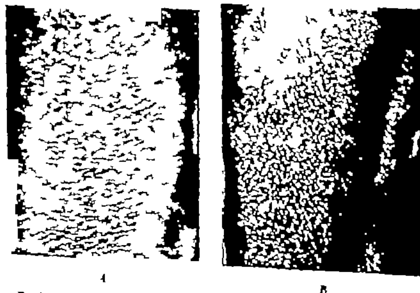


Fig. 369.—Better healing by cignolin in petrolatum (A) than by cignolin in lanolin (B).

sorption and healing effect on skin diseases may act inversely It is possible that agents which are too easily absorbed pass through the skin too rapidly to be able to exert a proper effect. Finally another *important and many sided* problem is presented by the *combination* of different drugs in ointments and other vehicles Usually this question is given little consideration One simply mixes the various drugs as one sees fit *in the particular case.* One should however give thought to the simple fact that two chemical agents may act on each other in four different ways, not counting the staining reactions

1 Combinations may *enhance irritations* as *for instance* by *mixing iodine* and mercury Unguentum Rochardi which contains iodine and mild mercurous chloride (calomel) is used as a strong skin irritant It is less generally known that *salicylic acid in higher concentrations combined with ammoniated mercu*

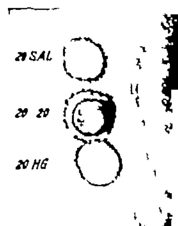


Fig. 370.—Bullous irritation by combination of salicylic acid and ammoniated mercury

ry causes severe irritations because the two agents *together produce mercury* bichloride (sublimate) (Fig 370) This is of practical importance because salicylic acid as has been mentioned before on account of its keratolytic quality is generally used to enhance *the effect of other drugs Even without the produc*tion of new compounds salicylic acid increases the irritative property of many medicaments such as sublimate resorcinol and chrysarobin probably because *it damages the horny layer*

2 Combinations may weaken irritative effects. Occasionally this may be caused by the addition of one single medicament to a vehicle In one of my patients, *petrolatum and lard caused weeping irritations* which regularly failed to appear if 2 per cent boric acid was added to the ointments (Fig 371) Chrysarobin ointments lose their irritative effect completely on the addition of alkaline soap because it inactivates chrysarobin

3 Combinations may *enhance the healing effect* The best known and practically important example is the combination of the various antieczematics especially *sulfur and tar* with salicylic acid (Fig 372) Occasionally an inten

sification of effect may be accomplished by using two drugs alternately e.g peeling first with salicylic acid, then applying tar then again salicylic acid etc.

4 Combinations may *weaken the healing effect* or even abolish it entirely For example, the addition of soap to chrysarobin abolishes not only its irritative effect but also its healing effect (Fig 373)

Naturally influences on irritative and healing effects need not always run in the same direction. For instance, chrysarobin in collodion irritates psoriasis more often but heals it less often than in paste. The combination of chrysarobin with salicylic acid significantly enhances the irritative effect of chrysarobin but has relatively little bearing on its healing effect.

These problems are multiplied if we start to work with triple or *multiple combinations* For instance, the addition of soap to chrysarobin abolishes the healing effect of this agent as has been stated before However the addition of salicylic acid to the chrysarobin-soap-ointment preserves the chrysarobin healing effect. This is the principle of the Dreuw s psoriasis ointment which is much used in Europe. If one should get the notion to omit the salicylic acid from this salve, e.g because of a hypersensitivity to salicylic acid one would use a completely ineffective product. Though it is true that the addition of salicylic acid preserves the effectiveness of the soap-chrysarobin combination, it does not reach the full healing power of the original chrysarobin paste (Fig 374) Therefore, the ingeniously composed Dreuw s ointment i.e. the soap-salicylic acid-chrysarobin paste is inferior to the simple chrysarobin paste in several ways. Another disadvantage of Dreuw's ointment is the fact that the addition of soap and salicylic acid creates an impediment to *immunisation* Several times I had the opportunity of observing psoriatics who showed recurrences on the Dreuw's ointment-treated side, while the other side, which had been treated with simple chrysarobin-tar ointment remained free (Fig. 375) This demonstrates clearly the danger of therapeutic failures inherent in too liberal a use of combinations. The combination of drugs is a responsible matter and it is the task of dermatotherapy to investigate the effect of each combination separately The dermatologists who mix their drugs in an undisciplined way will finally wind up with those arm-long medieval combinations which have aptly been called shot-gun prescriptions in allusion to the poor marksman who hopes that at least one of the many grains of shot in his blast will hit the target. Now we have seen that in treatment with effective and chemically aggressive substances the grains of shot do not even always remain shot. The larger the number of combinations in an ointment, the closer one gets to quackery i.e., treatment with therapeutic measures the effect of which is difficult or impossible to predict.

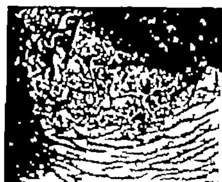
In *summarizing the external treatment* of skin diseases, the procedure runs as follows (1) preliminary observation preceding the start of treatment, (2) bland starter treatment with vehicles only and simultaneously (3) patch testing with chemically active medications, and (4) treatment with such chemotherapeuti-





A B

Fig 371 —Prevention of irritation from an ointment (A) by addition of boric acid (B)





A B

Fig 372.—The healing effect of chrysarobin (A) is strengthened by combination with salicylic acid (B)



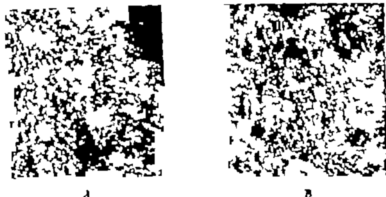


A B

Fig. 373.—Healing from chrysarobin (A) is prevented on the other side by combination with

cally active medications. This phase starts with mild medicaments in weak concentrations and, if possible, with the drug which offers the greatest likelihood of a cure. As long as healing progresses, the treatment is not changed. Never change a winning team! If the healing effect is unsatisfactory but the drug well tolerated the concentration is increased relentlessly up to the possible limit. Finally by the proper use of *combinations* intensification of therapeutic effect is brought to its climax.

It goes without saying that this type of treatment does not rule out the simultaneous application of other tried methods such as radiation therapy internal treatment and surgical intervention In many cases it is even indispensable to use these methods.



A *B*

FIG 174 —The healing effect of chrysarobin (*A*) is weakened by combination with soap and salicylic acid (*B*) as compared with other side (*A*) which was treated with chrysarobin paste only

It is easy to gather from the preceding that this type of treatment is always geared to a certain *medicament* which is at first brought to maximal concentration and finally combined with others one at a time. No matter how logically founded the procedure may appear this is not the type of therapy which is generally practiced. Much rather it is a widespread custom to treat with "package prescriptions," i.e. combinations of various bases with various medicaments which have been recommended by someone somewhere, and which are passed on from textbook to textbook and from clinic to clinic (e.g. Lassar's paste, Arning's tincture, Dreuw's ointment, etc.) This custom has some important disadvantages. These ready-made prescriptions frequently contain agents the reason for the inclusion of which no one knows, as for instance, salicylic acid in Lassar's paste. By treating with such prescriptions the physician becomes habituated to blindly following the words of teachers or of authors. This is a very antiquated method. The physician in everything he does should know well

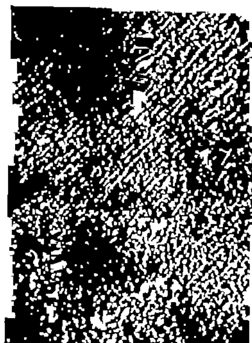




Fig. 371.—Prevention of irritation from an ointment (A) by addition of boric acid (B).





Fig. 372.—The healing effect of chrysarobin (A) is strengthened by combination with salicylic acid (B).





Fig. 373.—Healing from chrysarobin (A) is prevented on the other side by combination with soap (B).

cases, we have the task of omitting one ingredient after the other whether theoretically well founded or not and testing in the right left experiment whether the rump prescription" thus obtained does not have the same effect as or even a better one than the original. As has been demonstrated with the example of Dreuw's ointment, it can be expected that by following this course we shall be able to treat with much simpler and more lucid prescriptions.

It is not only that the misuse of package prescriptions misleads us into slipshod work by accustoming us to treatment with mysterious additives and empirically uninvestigated combinations, but it also has the great disadvantage that it prevents us from using our effective medicaments in *maximal strength*. It is therefore a source of insufficient therapeutic success, because ready-made prescriptions also have the peculiarity of calling for certain percentages of the effective drugs. This is frequently a narrow range which stops at a certain concentration (e.g. 2–10 per cent). This is wrong. If we are in the possession of an effective medicament for a certain disease then we must try to apply it in full strength. The limit should not be drawn by the recommendation of some textbook author but rather entirely by the qualities of the drug which make a higher concentration impossible (odor staining power caustic or other toxic effects, impossibility of incorporation into a suitable vehicle). The limits of this maximal application in the various vehicles should be stressed in the textbooks, including particularly the statement as to whether the agent can be used "pure" (undiluted). This is much more important than the notes on official percentages. There are hospitals where the entry in the records simply reads tar paste," since this combination is never prescribed in other than 2 per cent concentration.

We must steer clear of the suggestive influence of the prescriptions with which we have grown up. The treatment with package prescriptions, which is a remnant of the magic medicine of the Middle Ages must be quite generally replaced by treatment with effective remedies." We should refuse to use combinations the therapeutic significance of which is unknown because the inventor or the transmitter does not divulge its composition. We should refuse to use compositions of drugs whose effects have not been adequately tested by clinical experiment individually as well as in combination. And we should also refuse to be pressed into procrustean percentages if we have reason to believe that they fall short of the possible maximal application and healing effect of the agent. In doing this, we shall not only educate ourselves to a higher sense of therapeutic responsibility but also give better service to our patients.

why he is doing it. Treatment with such ready-made prescriptions lulls to sleep the critical viewpoint and sense of responsibility which the therapist should have. It demoralizes his medical conscience. Therefore, treatment by package prescriptions is a bad habit which must stop. *We must devote our wholehearted interest to effective therapy.* Whoever is profoundly interested in educating the younger generation of physicians to a really scientific way of therapeutic thinking *must sharply condemn* the blind passing-on of prescriptions which are not *in all details* founded on fact.

Of course, the dermatologist still needs a certain number of prescription formulas, namely, for *vehicles*. But this is another story. Here we are dealing with



Fig. 375.—Prevention of immunization by combination of chrysarobin with soap and salicylic acid (Dreuw's ointment): recurrence on the side treated with Dreuw's ointment only (B); no recurrence on other side (A), which was treated with chrysarobin-tar-lanolin.

chemically inert substances which must be mixed in certain proportions because they would otherwise not get the particular consistencies which are the reasons for their use. This, however, should not mislead the physician into thinking in terms of package prescriptions with regard to chemically active substances and to accept *traditional combinations and mysterious additions* without opposition or criticism. We no longer need to do this. Since the extraordinary efficiency of the *one side method* for the study of our external therapy has been discovered and put into a proper light, the possibility exists *of investigating accurately* the healing power of each medicament with this clinical experimental method unless its healing value is already obvious. This is especially necessary in all *package prescription formulas* which history has handed down to us. *In these*

In *short-wave* diathermy still higher frequencies (10–100 million per second) are used with wave lengths of 100–10 meters or less. Such very short waves are used for deep diathermy and therefore generally have no dermatologic application.

HEAT TREATMENT

In dermatology heat treatment is most frequently carried out by the *closed (impermeable) wet dressing* (Priessnitz dressing) which has already been discussed (p 206) By the addition of a source of heat to the moist dressing (hot linseed poultices, heating pad) one creates a steam *vapor dressing* *Dry heat* alone (heating pad hot air diathermy short waves) is less used in dermatology and is probably less effective. Moist-heat treatment carries the disadvantage that it macerates the skin which may cause irritations and favor infections. If the skin is irritable (eczemas) impermeable dressing should not be used, and in infectious processes (pyodermas mycoses) it is necessary to use disinfectant solutions (Burow's solution silver nitrate) and cover the surrounding areas with pastes or place the moist cloth over a layer of antiseptic ointment (20 per cent ammoniated mercury in petrolatum 33 per cent sulfur in petrolatum) The most important indications for this type of dressing are deep trichophytosis, painful boils, paronychias, and lymphangitis Simultaneous systemic treatment with sulfonamides or other antibiotics should be administered if it seems indicated.

Very short applications of heat by *dabbing with hot water* are effective in intertriginous eczemas, but mostly in these only They may also be used for transient relief of itching in non-eczematous skin diseases. In intertriginous eczemas, one simply presses a dripping wet sponge or folded washcloth three times against the affected area Then an ointment is applied The water should be as hot as possible since hot water is better tolerated than cold In some cases of pruritus, lukewarm applications have a better effect than hot or cold ones.

Another form of treatment with moist heat is *steaming* which is utilized for the support of acne treatment. It is most simply done by holding the face over a pot of boiling water covering head and pot with a towel. There are steaming apparatuses with large glass bell jars designed to facilitate the treatment. One should not expect too much of these devices.

Hot-air apparatuses are occasionally used for the treatment of chronic circulatory disorders. In the same conditions, *massage* is also used. I shall discuss this therapy here because, like heat it produces a deeper-reaching hyperemia and generally has a stimulating effect on the circulation. Therefore massage is indicated in chilblains, chronic edema and elephantiasis keloids, scars, and scleroderma. It is of greater cosmetic importance for the removal of localized accumulations of fat and wrinkles, though obviously with only limited success. Massage is always done with oil or vaseline. The direction of the massage movement is of importance, particularly in the face On the forehead eyes and

CHAPTER ELEVEN

Physical Therapy

OUR methods of physical therapy essentially involve the therapeutic use of *electromagnetic radiations*. The following list indicates the respective wave lengths

Direct (galvanic) current (d.c.)	
Alternating current of low frequency (faradic current)	5 000–1 000 km.
Alternating current of high frequency (diathermy)	600–6 meters
Heat rays including infrared radiation	0.4–0.0007 mm.
Visible light	7 000–4 000 A[1]
Ultraviolet radiation	4 000–40 A
Grenz rays (Bucky rays)	2.5–1.5 A
X rays	1.0–0.1 A
Gamma rays of radium	0.3–0.0006 A

THE USE OF DIRECT CURRENT AND ALTERNATING CURRENT

The *galvanic current* is little used in dermatotherapy except for some minor surgical procedures. It has also been used in the method of *iontophoresis* by which one tries to introduce certain medicaments into the deeper layers of the skin (ichthyol iodine cocaine histamine) but the results have mostly remained unsatisfactory

Electrolysis which is based on the caustic effect of alkalis acids and salts which form when the galvanic current passes from metallic to tissue liquid conductors plays a part in dermatologic surgery It will be discussed later

The use of *alternating currents* has more varied applications

In an obsolete form of *high frequency treatment* the skin was stimulated by sparks from electrified evacuated glass tubes This type of treatment was used for alopecia and warts but had little or only placebo value.

Diathermy is of greater importance Here the frequency of the alternating current is so high (± 1 000 000 per second) that the electrifying sensation ceases. At the same time there is generation of heat in the tissue By the use of suitable electrodes, this generation of heat can be so vigorous that the tissues coagulate and can be cut with a wire electrode without capillary bleeding By using a wire loop one can remove the skin in layers. This method will be discussed with the surgical operations.

1 A = 1 Angstrom unit = 10^{-8} cm

scattered small foci of lupus erythematosus, though one must start with very short initial treatment times (1 second) because of the danger of systemic exacerbation, which may be provoked in this disease by any type of treatment.

If used correctly freezing may spare the patient long-drawn-out series of injections or courses of internal medication. Higher doses (60 seconds and more) may and should be applied in ordinary warts (verrucae vulgares) However here the method is too time-consuming if there are numerous warts. It is also difficult to apply the correct dose because it must vary according to the thickness of the wart and its horny layer Therefore the results are unequal, but it has been claimed that they can be improved by X-ray or radium treatment immediately following the freezing a procedure which is also supposed to be successful in corns. Flat and seborrheic warts and senile keratoses are more suitable, but it is necessary to remove the thick keratosis first. The most vigorous freezing is required by epitheliomas but only the very superficial forms are suitable for such treatment. Such vigorous treatment is followed by necrosis, which, of course, heals with atrophy This, however is no objection because the patients are mostly elderly people and the other available methods (surgery X-ray radium) also leave atrophies and scars. A most effective method of intense cold application is the use of *liquid nitrogen*. This is an otherwise little-used and therefore inexpensive by-product from the manufacture of oxygen It has a boiling point of −195 78° C Skin freezes almost immediately when a cotton stick applicator dipped in liquid nitrogen is pressed against it. Warts need about 20 seconds (two to three applicators) There is some pain when the freezing tissue reaches a certain temperature, but the pain stops when the tissue is thoroughly frozen. When the frozen tissue thaws again the pain recurs for a short time. If applied correctly to a wart the resulting blister lifts the wart up so that after a few days it can easily and without any pain be cut away with scissors. It is a good method for removing *multiple* warts on the hands The liquid nitrogen is supplied and transported in a vacuum (Thermos) bottle with a perforated cork stopper which prevents spilling as well as the building-up of pressure in the bottle. A fresh supply of 1 liter of liquid nitrogen will keep 2–3 days at room temperature before it has completely evaporated

A short freezing of the skin can be accomplished by *spraying with ethyl chloride* The method has been recommended for nevus flammeus, alopecia areata psoriasis, and lupus erythematosus, but it does not seem to be very promising It is certainly ineffective and is therefore objectionable in nevus flammeus

LIGHT TREATMENT

In contrast to X-rays the rays of visible *light* with the longest wave lengths have the greatest power of penetration. Therefore, the red rays penetrate deepest the blue-violet rays are mostly absorbed after penetrating 1 *mm.* and

upper lip the movements run sideward on the nose and cheeks, downward and upward and on temples and chin downward

COLD TREATMENT

Intensive application of cold is accomplished with *dry ice* (solid CO_2) Carbon dioxide snow is molded into pencil sticks in tubular forms and then under pressure applied to the skin Pressing the dry ice against the skin results in a frozen white disk which thaws again *in a few minutes and is followed by* an erythematous wheal or after more vigorous application of the dry ice, by a blister The floor of the bulla is superficially necrotic. The pain is bearable The effect *depends on the pressure and the time of application* If one always tries to apply the same medium pressure one may dose the treatment with sufficient accuracy by measuring the time Cautious freezing starts with 3 seconds and *according to the reaction* the time is increased systematically every *1–2 weeks*. Since local tolerance develops, an increase in the treatment time becomes necessary The counting of the number of seconds is much facilitated by the use of a metronome since it is inconvenient to look at a watch while treating Since hair even fine lanugo greatly impedes the effect of the cold shaving is often necessary The skin of the face and the flexures is more sensitive than the skin of the extensor aspects The skin of infants is particularly sensitive. After only 4 seconds of dry ice treatment applied to the face of a four year-old boy I saw a slight atrophy develop

Treatment with dry ice is fraught with much greater cosmetic danger than is expressed in textbooks. The depigmented and anemic white spots and atrophies which remain are extremely unsightly and conspicuous because they exhibit the circular or angular form of the dry ice stick which has been applied *under pressure* This *is most embarrassing* when the blanching has taken place in a telangiectatic area (vascular nevi) and red stripes still remain between the blanched areas, an imperfection which cannot be completely avoided even with *angular forms* If *the treatment* has caused glossy and wrinkled atrophy the poor cosmetic result is still worse Therefore treatment with dry ice is a matter of great responsibility

The most important indication for dry ice treatment is given by some vascular and some pigmented nevi Among the vascular nevi only the small elevated angiomas are suitable The more extensive and especially the flat nevi flammei *(port wine stains) never blanch evenly* but instead result in exceedingly ugly mottled disfigurations. Dry ice treatment of these lesions is therefore contraindicated For lentigines on the face and for vascular spiders, the procedure is a *cosmetically* questionable one because of the white circles which may remain On the other hand some raised pigmented moles respond better to dry ice than to the microburner (microbrenner needle hot cautery) The results in keloids and xanthomas are unpredictable Dry ice is very suitable for

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which, in a given patient, elicits an erythema with subsequent desquamation without causing a more marked and uncomfortable burn. Since the skin becomes accustomed to the radiation, it is necessary before every radiation to inquire about the reaction and to step up the time if a reaction fails to appear. In this way one will soon arrive at an exposure to which the patient no longer becomes tolerant but responds with the same degree of erythematous reaction to every treatment of the same length of time. With this method it is, of course, necessary that the distance which has been chosen be kept constant. This makes it imperative to measure accurately the distance from the source of light, since the intensity of the radiation varies inversely to the square of the distance, so that minor variations may result in significant changes of dosage. Therefore, the lamp must be equipped with a firmly attached device for measuring the distance. Measuring with a metal or wooden rod held in the hand is unsatisfactory. A second prerequisite is that the time interval between the radiations be kept constant, since after the reaction the acquired tolerance to light decreases with every day. If the intervals are too long, the reactions would become too strong, and if they are too short, the reactions would be too weak. It is therefore advisable to treat skin diseases with ultraviolet rays only once a week. More frequent exposures cause heavier pigmentation and thickening of the horny layer, making reactions more and more difficult to elicit. It also is of importance to advise the patient not to expose himself to the sun unnecessarily, since increased tolerance might prevent the desired radiation reaction. If, however, the pigmentation and horny layer thickening rather than the erythema are considered to be the healing factors, it is necessary to treat at short intervals, daily or every second day. In this case it is also advisable to use a carbon-arc lamp either alone or in combination with the quartz light, since this type of light has a stronger tanning effect.

Frequently the indications for the ultraviolet treatment have been set up too vaguely. A substantial improvement can be expected mainly in acne, rosacea, various forms of alopecia, chilblains, and tuberculids. Acne and rosacea are mostly much better cured with salves than with this time-consuming method. In alopecia also it is better first to try the tinctures, which can be applied so much more easily. In ordinary premature male type baldness, the Alpine Lamp has no effect at all, much to the amazement of the laity. In lupus erythematosus I must warn against ultraviolet treatment, because more vigorous reactions cannot always be avoided in view of the difficulties of a really exact dosage. Such reactions may cause systemic exacerbations and even death.

A mercury vapor lamp which makes possible the irradiation of the whole body is the Jesionek lamp. The total exposures are best accomplished with an arrangement of four lamps. The lamps are placed in a square, within which the patients walk around on a circle painted on the floor, changing direction after each completed circle. With this treatment, it is also necessary to start cautious-

the ultraviolet rays after only 0 1 mm of depth Despite this fact it is just the superficially absorbed rays which exert the strongest effects on the skin and therefore are used predominantly for treatment purposes.

Long wave light rays are applied with infrared bulb type lamps of which there are several on the market Their effects seem to be essentially caused by heat Therefore treatment with such lamps has little importance It is used for boils and poorly healing ulcers.

Several lamps are in use for the utilization of the short wave part of the sunlight spectrum Natural *sun baths are impractical for the purpose of dermatotherapy* though many people think that the sun is a panacea to which they expose themselves with enthusiasm especially when they are afflicted with a skin disease *Unfortunately* the physician concerned with the welfare of his patients must deprive them of this beautiful delusion Sun bathing is not entirely innocuous even for a healthy person because it is so difficult to find the necessary limit Sunburn lassitude headache and nervousness are the common effects of excessive exposure *For many patients suffering from skin* diseases, sun worship is actually disastrous because most eczemas do not tolerate sun at all and in some diseases excessive sun treatment may lead to impairment (psoriasis) scars (hydroa xeroderma) or even life threatening exacerbations (lupus erythematosus) Even in those dermatoses which may improve by exposure to the sun (acne rosacea alopecia) this effect mostly fails to materialize because the intensity of the sunlight in many areas is too weak and inconstant Since in these diseases, a good healing effect can be expected only from an erythematous reaction *nothing* can be expected from natural sun radiation since it does not permit careful dosage As is so often the case here too much to the chagrin of all serious lovers of nature the artificial healing procedure with ultraviolet lamps is often much superior to the ordinarily available natural sunlight

Treatment with ultraviolet *rays from artificial sources* can be done as *radiation from a distance* or as close-contact radiation in the form of *compression radiation* For *radiation from a distance* mercury vapor lamps are mainly used for limited areas (*face hands solitary ulcers*) *for this the Alpine Lamp* type is suitable The prerequisite for good effect is exact dosage which however cannot be given in definite numbers because we have to consider two extremely variable factors. These are *the strength of the lamp which decreases* with the number of hours used and the sensitivity of the patient which not only varies from individual to individual but also depends on his previous exposure to light and therefore on the season *Measurements with the available dosimeters* are therefore unnecessary One simply starts with the shortest distance which the size of the field and the heat of the lamp permit (usually 30 cm) and with an estimated short treatment time (½–2 minutes) which is *then gradually* increased. *In* this way one quickly succeeds in finding the time of radiation

GRENZ RAY (BUCKY RAY) X RAY AND RADIUM TREATMENT

X-ray treatment is such an important and responsibility-carrying therapeutic method that every dermatologist must be familiar with its essential indications and risks in the treatment of skin diseases.

Compared with external dermatologic treatment, X ray treatment has the obvious advantage that it saves months of time-consuming applications of salves and dressings and does not cause staining or odor It acts faster and is infinitely more convenient Furthermore, it sometimes helps in cases which are resistant to all chemical means. On the other hand however it can be used without difficulty only in circumscribed conditions and is very dangerous in inexperienced and careless hands. Another disadvantage is that it cannot be freely stepped up or repeated if the effect is unsatisfactory

In contrast to ultraviolet-ray treatment a reaction should be avoided with X ray therapy in most cases. Such reaction is an X-ray induced erythema which takes a wavelike course. It appears on the third fourteenth, and fortieth days, though with considerable fluctuations. Individual differences in sensitivity of such magnitude that they have to be considered in treating various patients do not occur Systemic reactions called *X-ray sickness* which frequently are dreaded by patients are not ordinarily encountered because of the topical and superficial character of dermatologic radiation therapy In stronger reactions, a bullous X-ray dermatitis may occur But the greatest danger lies in the fact that, even without preceding early reaction serious late sequels may occur after months years, or decades. These late changes are atrophic and sclerotic X ray scars which because of hyperpigmentation, depigmentation and telangiectases, are most conspicuous, ugly and in some cases even severely crippling X ray ulcers may resist all attempts to induce healing Verrucous X-ray keratoses and desquamations which are completely resistant to therapy may become a great impediment by their dryness and painful fissuring. Finally X-ray carcinomas may develop in ulcerations and keratoses and endanger the patient s life by their malignant growth. To protect patients from such consequences, many safety measures are necessary

The first prerequisite of responsible X-ray treatment is proper *ray and shock proof* equipment. The second requirement is exact measurement of dosage. The dosage must be measured ionometrically so that it can be expressed in international X-ray units (r) and the output from the machine must be checked at regular intervals. During X-ray treatment the patient s position must be carefully adjusted to permit perpendicular irradiation. The healthy surrounding skin must be covered with suitable pieces of sheet lead. The same must be done with especially sensitive regions, such as hairy areas, vermilion borders of the lips testicles, and eyes, the latter because of the danger of cataract formation as a late sequel. Overlapping of the fields of radiation must be avoided. Correct centering of the field must be ascertained Accurate measurement of the dis-

ly and to increase the time of exposure regularly Some light therapists who think that pigmentation is more important than the erythema add a carbon arc lamp over the center of the circle In the past, this type of light bath had a limited though important indication namely tuberculosis luposa It seemed that the topical methods of treatment had a better effect when combined with radiations of the entire body which was explained by a stimulation of biological defenses through the light treatment. The diseased skin areas are covered in the ambulatory light bath while the total light bath is given Since the advent of vitamin D_2 and even more recently effective antibiotic treatment of lupus vulgaris the light bath has lost the greater part of its importance

Compression radiation with ultraviolet rays makes a deeper effect of the radiation possible. For medium-strength compression radiations, the *Kromayer lamp* is suitable This is a mercury vapor lamp with a quartz window which is separated from the source of light by a cooling water jacket The quartz window therefore does not get hot and can be put directly on the skin The indications are principally the same as those for the Alpine Lamp but of course only small areas not exceeding the 3-cm diameter of the window can be treated[2]

The strongest compression radiation effect is accomplished with the *Finsen lamp* which furnishes a carbon-arc light which is condensed by a water-cooled system of quartz lenses The field of radiation is still smaller than that of the Kromayer lamp (1 cm in diameter) and the reaction to a correctly executed irradiation is always a blister with a superficial necrosis at its floor Nevertheless even with the worst reactions only very fine cosmetically satisfactory scars remain

The original Finsen lamp was a bulky apparatus with four tubes to treat four patients simultaneously Because of its high cost many were satisfied with the simpler Finsen Rheyn lamp with which only one patient could be treated at a time. However the results of this arrangement were less reliable An improvement over the Finsen lamp was the Lomholt lamp which required shorter treatment times

There was essentially only *one* indication for the Finsen treatment namely tuberculosis luposa The great significance of the method consisted in its excellent cosmetic results. Sometimes the scars were hardly visible The disadvantage however was that it required indefinite time and patience Because of the small size of the fields and the long treatment times per field the patients usually were required to spend the whole day under the lamp from 9 00 A.M to 6 00 P.M with time out for lunch and this torture had to be repeated every week for months and years. The advent of successful treatment with effective antituberculosis drugs meant relief for these patients and the Finsen lamp lost its importance.

2. I recent years the water-cooled model has been largely replaced by an air-cooled on hich however does not permit compression.

some weeks after the treatment, the patient so to speak lives "beyond his means. In diseases with a great tendency to recur such as psoriasis, the doctor may get into a difficult situation because the patient who has once experienced the convenience of the salveless treatment is likely to press for repeated radiations. One should be on guard not to yield to such requests, once the limit of the non-dangerous dosage has been reached. For this reason it is advisable to be very reluctant in the first place to use X-ray treatment for usually recurrent diseases. It has happened not rarely and I have seen it repeatedly that psoriatics have died pitifully from X ray cancers. One should also be conservative with the use of X-ray treatment in diseases which have a less marked tendency to recur for example, chronic eczemas. In the early days of the X-ray era it was customary to subject such cases to X ray treatment without hesitation When the disease then recurred it was much more difficult to get the patient to submit to the disagreeable external treatment than it was in the beginning For this reason alone it is always better to start with ointment treatment and to keep X-ray treatment in reserve for those residual symptoms which fail to yield to chemical agents. What is more important in this way much more lasting results are obtained Thus X-ray treatment belongs at the end of the eczema treatment program and not at its beginning This also has the advantage that the irritative and curative effects of the most important medicament can be investigated beforehand, so that one can combine radiation and topical drug therapy without fear of unexpected irritations. Furthermore this plan makes it possible to prescribe a suitable after treatment for additional protection against recurrences The doubts which were formerly expressed against the *simultaneous* administration of ointments and X rays are not justified. On the contrary the X ray treatment of superficial chronic skin diseases *should* be combined with the application of ointments. Therefore, superficial X ray treatment does not belong in the hands of the roentgenologist who is not equipped and frequently not much inclined to carry out ointment treatment properly Its only place is with the dermatologist. By the way comprehensive roentgenologic training of the dermatologist is not required since compared with deep X-ray therapy superficial radiation is a simple procedure which, in many dermatologic clinics, is carried out by technicians and nurses.

The most important *indication* for soft to medium-hard X-rays are the chronic papular lichenified, and other types of eczemas Psoriasis and circumscribed foci of lichen planus also frequently respond well In psoriasis, however one should for the reasons given before, treat only resistant residual patches or occasionally psoriasis of the nails because the latter does not respond well enough to external treatment. Unfortunately it also frequently fails to respond to X rays. In some rare and recalcitrant skin diseases, such as folliculitis barbae and mycosis fungoides, X-ray treatment is of great importance and also as a last resort in a variety of inflammatory processes without certain diagnosis. In

tance of course must be provided by a suitable measuring device, and the exposure time must be checked by a reliable timer or an attendant with a stop watch Patient and apparatus should never be left alone Finally treatment dosage must be expertly predetermined *and if possible administered in fractions* The administered doses must be entered on a suitable record sheet with careful *indication of the treated skin fields* so that additional treatment of already amply irradiated skin can be avoided with certainty The therapist who *fails* to pay conscientious attention to all these precautions is guilty of negligence toward his patient and damages the good reputation of a valuable treatment method

Not all cells are equally sensitive to X rays and so these rays act selectively Cells which divide rapidly such as carcinoma cells the basal cells of the hair papilla and also the vascular endothelium are more heavily damaged than the rest of the skin This explains the success of X rays in carcinomas and some other neoplasms and also the possibility of X ray epilation Since hard X-rays with short wave lengths penetrate more deeply into the tissue than do soft rays it is possible to concentrate the strongest effect on certain layers of the skin by the use of aluminum and copper filters. Therefore, in more deeply seated lesions such as abscesses and many carcinomas the use of such filters is indicated

In such superficial skin diseases as *eczemas, soft to medium hard rays* of up to 80 kv unfiltered or with as little as ½–1 mm of aluminum filtration are used. *Generally no more than 100–150 r per field are given in one treatment*, and this dose is repeated if necessary three to four times in intervals of about 10 days, so *that the total dose in one series does not exceed 400–450 r This is called the eczema dose* Fractionation has the advantage that if the response is good only a part of the total dose need be given This is a definite advantage in case later radiations should become necessary If the prognosis is doubtful it may be advisable to give the whole series to be better able to judge later how far one may venture to go Such a series may be repeated without risk after 3 months. A second repetition should not be done before a year has passed Further repetitions should be carried out only with great reluctance since skin which has once been radiated remains more sensitive for life and is more susceptible to X ray damage than skin which has never been treated It is for this reason that one should never radiate unless absolutely necessary or treat too large fields because one may deprive one s self of the possibility of treating subsequent skin diseases with radiation On the hairy scalp the full series must not be given because it is generally followed by a transient falling of hair Here two to three treatments are enough Since X rays have no preventive effect, prophylactic radiation of normal skin is not permissible

X ray treatment has the great *disadvantage* that in many cases it suppresses the disease only temporarily the symptoms recurring after about 6 weeks. For

In diseases which extend deeper into the skin, one uses *harder* X rays by filtering with 3–4 mm. of aluminum and stepping up the kilovoltage. In such cases, it is general practice to give a total dose of 400–450 r in only two sittings or even not to fractionate at all. The most important indications for this type of deeper-surface therapy are abscesses (acne abscedens, hydradenitis axillaris, tuberculosis colliquativa, boils, and dermatoses with marked thickening of the horny layer such as tuberculosis verrucosa, verruca plantaris)

Still harder rays (0.5-mm. copper filter) are used for the treatment of neoplasms, especially epitheliomas. The seriousness of these conditions and in most cases their relatively small size permit and require high doses which entail X-ray atrophy

Still more suitable in epitheliomas of small size is the application of very high doses of soft rays in small fields, mostly not exceeding 3 cm. in diameter This method is called *contact radiation* Chaoul radiation or X-ray caustic therapy because 1 2 weeks after the radiation necrosis and sloughing of the uppermost layers of the skin regularly occur It is best to fractionate with daily doses of 500 r until the inflammatory reaction starts, but the treatment may also be given in one dose of 3,000–4 000 r which may be repeated after weeks or months if the result is unsatisfactory X ray caustic therapy is frequently a good substitute for radium treatment though special equipment is necessary for good results.

Besides the relatively soft alpha and beta rays, radium also emits gamma rays which exceed in hardness even the hardest X rays. In order to utilize these rays alone, brass filters are used. The radium is encased in capsules or plaques[3] which are attached to the skin with adhesive tape or in needles which are inserted into the tumor (interstitial radiation) *Mesothorium* is used in a similar manner but it does not possess such a high percentage of hard gamma rays as does radium. The dosage of radium is commonly given in milligram hours based on the content of radium element in the preparation used. Good results *with* radium treatment can be expected only if relatively large amounts of radium are available. Radium treatment and hard X-rays are indicated mainly in epitheliomas, growing angiomas and keloids. In hypertrophic scars and Peyronie's disease (plastic induration of the penis) radium is superior to X rays. In larger nevi, including nevi flammei, it should not be tried because the customary small fields of application result in the same exceedingly ugly patterns as those encountered with dry-ice treatment. It is never possible to make the fields accurately adjoin without any interspaces. Therefore, a telangiectatic network remains in which are set the dead-white often even depressed fields of radiation. The picture is even uglier than after dry ice not

3. The radium preparations which are most frequently used in dermatology are plaques containing 10–30 mg. of radium spread evenly over areas of 1 12 sq. cm. These plaques have 0.1-mm. monel metal filter which also allows much of the beta radiation to pass through. A so-called full-strength plaque contains 10 mg. of radium spread over sq. cm.

acne it is better not to use radiation except in abscessed and keloid-forming types. Acne usually does not respond to X rays and can be sufficiently improved by methodic external treatment. In general it is better not to apply X rays in diseases which can be successfully treated with innocuous means, except in special cases. Twice I have seen patients *one of them a piano teacher whose* hands had been terribly mutilated by radiation which was intended only to *remove a few harmless warts! It is important to know that X rays have no* effect whatsoever on most stationary skin anomalies such as all kinds of birthmarks including port wine stains ichthyosis keratosis, etc.

On the other hand X ray treatment has become a real blessing in the treatment of the mycoses of hairy areas especially tinea barbae and tinea capitis in children These fungus infections especially the relatively non-inflammatory types, usually will heal only if all hair is removed prior to the application of disinfecting ointments. This is not possible by manual epilation and the old method of epilation with pitch plaster is torture However by correctly applied X rays a *temporary epilation* can be accomplished without pain This requires the application of a single dose of 375 r (so-called *epilation dose*) In the bearded area four fields on the scalp five fields which have to be centered conscientiously are treated The border lines between the fields are not covered because on account of the convexity of the radiated areas the peripheral parts of the fields would receive too small a dose if the fields did not overlap Errors or inaccuracies in technique *may result in atrophic spots* which remain hairless for life and which represent a permanent accusation of the therapist About 3 *weeks after proper radiation the hairs become so loose that most* fall out spontaneously The remainder must be pulled out with tweezers or adhesive tape a procedure which *in this stage is easy and painless In a short time the entire* scalp is bald and glossy like a billiard ball Simultaneously the disinfecting ointment treatment is started and continued for *several months As the hair* grows back the cure is checked by several examinations for fungi

Peculiarly on the bald skin in alopecia areata the epilation dose *may have a* paradoxical effect namely apparently to cause restoration of the growth of hair on the hairless areas The same effect can be elicited with *grenz rays* (*see* below) The latter method is preferable because of the smaller risk and the possibility of repeated radiations

Of course attempts have been made to effect *permanent epilation* as treatment for localized hypertrichosis by increased doses of X rays. *However it has* been demonstrated that this is not possible without simultaneous severe X ray atrophy and damage to the skin Therefore it is necessary *to caution against* the treatment of hypertrichosis with X rays. The same is true of the treatment of localized hyperidrosis of the armpits and palms though *occasionally good* results have been attained without damage to the skin The procedure, nevertheless carries considerable risk

been treated previously with radium lighter areas caused by such treatment must be covered with barium paste to prevent dead white spots, which would jeopardize the entire cosmetic result. Unfortunately only some of the vascular nevi respond sufficiently to grenz rays the absolutely flat nevi flammei being more often resistant than the slightly elevated types.

The cosmetic risk of contact radiation is similar Here too atrophies may occur which may cause bald spots and X ray telangiectases on the scalp which are no longer amenable to any therapy In extensive vascular nevi this method permits more closely adjoining application of the small round fields than radium, provided that great skill and care are employed.

Similar to the grenz rays in the final result but much weaker is the radiation effect of thorium X, a radioactive preparation which is supplied in ointment alcohol, or lacquer vehicles. Application in lacquer form is preferable. The preparation must be fresh and applied immediately on arrival, since it loses its potency in a few days. The indications for thorium X are the same as those for grenz rays. It had been particularly recommended for nevi vasculosi and proved to be effective but too weak to gain practical significance. The introduction of the grenz rays has made it dispensable.

only because of the frequently depressed scars but also because in the white scarred fields as in X ray atrophy brown pigmented spots and dark-red branches of radiation telangiectasias appear which tend to make the appearance still more dappled and conspicuous In addition the power of facial expression may be disturbed by ugly contracting scars The treatment of more extensive birthmarks with radium should be refused and every patient should be cautioned against it

In contrast to the usual dermatologic soft X rays, *grenz rays* (Bucky rays) represent *extremely soft X rays* The German name meaning border line rays indicates that their longer wave length places them at the border between X rays and ultraviolet rays Because of their softness their effect is very superficial They are largely absorbed by various deposits such as scales crusts, ointments adhesive tape Therefore if necessary the field of radiation must be cleansed prior to radiation Grenz rays like X rays cause a reaction which takes its course in waves Usually 10 days after doses of 1 000 r or more an erythema or even a bullous dermatitis may develop This symptom ordinarily does not have the serious implications of X ray dermatitis but late sequelae may also be expected if the reactions are repeated After too frequent or too strong radiations grenz ray atrophy occurs, which has the same poikilodermic appearance (*hyperpigmentation depigmentation telangiectases*) as X ray atrophy and therefore is just as conspicuous and ugly

The indications for weaker grenz ray irradiations of 300–400 r several times at 2 week intervals are generally the same as those for soft X rays namely especially chronic eczemas However they are much less reliable A special indication is eczema of the edges of the eyelids because grenz rays cannot penetrate the lids to the cornea and therefore are innocuous to the eye In psoriasis the same considerations generally prevail as for X ray treatment Of course the risk connected with grenz rays is a much smaller one. But on the other hand grenz rays like ultraviolet rays occasionally cause provocation of psoriatic eruptions Grenz rays sometimes have a very favorable effect on Darier s disease In harmless dermatoses such as multiple warts they may be used with less reluctance than X rays because at cautious dosage the risk is smaller

On the whole the grenz rays represent a less dangerous, but also less effective substitute for X rays Their great significance lies in the fact that in many instances, we can by their help heal or at least improve an ailment which is almost completely refractory to all other methods of treatment including ultraviolet and X rays namely the nevus flammeus. It is true that grenz ray therapy of this condition requires rather high doses In general it is recommended that one give 1 000 r about six times in succession at 5-week intervals. Higher dosage carries the risk of telangiectases and freckle like pigmentations which may occasionally occur even at the dose described If parts of the nevus had

under block anesthesia For the correct execution of the operation suitable scissors, a raspatory dressing forceps, and curette are necessary The nail is first detached from its bed by inserting the raspatory from the free edge under the nail. Then the nail is cut longitudinally for its full length, and one half after the other is lifted out with the forceps by a rolling movement comparable to the detaching of the key strip from a can of sardines by turning the key Finally leftover cuticle on the nail wall is cut off and the nail bed is scraped as far as possible. The postoperative treatment must be done with disinfecting ointments and tinctures, but also the growing nail must be kept as short as possible with scissors and knife for a long time. Every week the corners must be cleaned out as long as fungi can be found. If physician and patient muster the necessary patience, it is frequently possible to heal onychomycosis though in most cases it will take years.

The *curette* is used mainly to remove warts, mollusca contagiosa and condylomata acuminata (venereal warts) By freezing with ethyl chloride, these tumors become harder than the surrounding skin so that they almost jump out like dry peas Just the right amount of tissue is removed by this method so that neither recurrences nor scars result. Thus, in this operation freezing not only has the purpose of anesthesia but is also required even if novocaine has been injected into the lesion

Within the last few years the treatment of pitted scars resulting from acne of the face, by *abrasive* methods, has become a successful dermatological procedure While the principle of grinding away small portions of skin with a fast rotating tool of the type used in dentistry had been known for half a century[1] the planing of large areas—for instance the entire face—has been developed only recently The skin is hardened and anesthetized by freezing with either ethyl chloride or dichlorotetrafluoroethane (Freon) Frigiderm is a Freon suitable for dermabrasion which comes in practical spray cans for ready use. It is non toxic if inhaled under practical working conditions, non-inflammable, and freezes more readily than ethyl chloride, which needs blowing and is highly toxic and inflammable The patient is prepared for the operation by an injection of 50–75 mg. of Demerol and the scarred areas may be prechilled with ice bags although this is not absolutely necessary The areas to be abraded are painted with 1 per cent gentian violet solution, to show the operator during the operation what has already been ground off The eyes of the patient are carefully protected against the freezing spray by greased, cup-shaped plastic spectacles. The hair is completely covered by a bathing cap. Gauze pads for the protection of the eyes are dangerous, since woven material may be caught by

1 Kromayer in Germany (1905) was the first to use a rotating tool, the dentist's drill, for the removal of blemishes but the method was practically forgotten except for biopsies. Schreus in Germany (1949) and Kurtin in America (1953) developed the modern tools and techniques. Wilson, [illegible] Luikart and Ayres, studied the histopathology of regeneration after dermabrasion and replaced ethyl chloride by Freon for freezing.

CHAPTER TWELVE

Minor Surgery in Skin Diseases

DERMATOLOGICAL surgery frequently tries to substitute for simple excision more complicated often multiple-stage methods which leave a scarcely visible irregularity of the skin instead of a linear excision scar In other words, the dermatologist aims at scarless surgery This cosmetic result depends mainly on the use of *small* instruments Therefore the ordinary *scalpel* rarely plays a role in the hands of the dermatologist Only occasionally it is used for lancing of abscesses In excisions for biopsy purposes, the knife is avoided and is replaced by the *tubular punch* (p 198) which if cylinders not exceeding 3 mm are used leaves only an inconspicuous pit. For scarification which some dermatologists do to remove branching telangiectases, sandpaper or a fine *lancet* is used Very superficial fine incisions are placed parallel and close to one another followed by a second identical row crossing the first one. The results of this procedure are often quite unsatisfactory I use a double-edged lancet for the excision of verrucae vulgares. Elevated common papular nevi are best cut off at skin level with a narrow *small curved knife* Then a pigmented plateau or at least a pigmented spot usually remains which requires after treatment with the microburner (see below) Sebaceous cysts are treated with the *knife needle* a needle-shaped very narrow blade which can be painlessly inserted into the extended mouth of the follicle and the follicle subcutaneously slit open This operation may be done once or if necessary repeatedly usually resulting in a scarring obliteration of the follicle without the slightest damage to the epidermis *A short strong knife with firm handle* is used to scrape off mycotic nails to improve the depth effect of disinfectant ointments and tinctures. Unfortunately this method in most cases is not sufficient and extraction of the nail must often be done

The *extraction of a nail* is a minor operation which every dermatologist should master since it is indispensable in onychomycoses. However one should not believe that the disease is eradicated by extraction of the nail. Just as in X ray epilation the extraction of the nail is only a preparation for the treatment proper with disinfecting medicaments This medicinal treatment must be executed with even greater care and patience because in contrast to the favorable results following epilation recurrences after extractions of nails unfortunately happen quite frequently The extraction of the nail is, of course done

Pitted acne scars are the main indication for surgical planing In this often disfiguring and distressing condition for which no other treatment is available dermabrasion has given good and sometimes excellent results. Its value for the removal of tattoos, keratoses, nevi and other benign conditions has not been sufficiently determined

An indispensable device used in the treatment of various skin anomalies is Unna s *microburner* (microbrenner) This is a platinum needle around which a heating wire is coiled, which, however leaves the point of the needle free The spiral heating wire is made red hot electrically while the needle itself does not glow but is only heated to a lesser degree. The microburner operates at a lower temperature than the thermocauter which it replaces in dermatologic therapy On tender skin as, for instance, on the face it leaves small pits which how ever may often even out in the course of a few months. It is necessary to use this tool very cautiously and without anesthesia if possible because the pain reaction of the patient is a good safeguard against too deep application. The microburner is particularly suitable for the removal of lentigines, small facial nevi and small fibromas, such as accompany tuberous scleroma of the brain (Pringle s disease) It may also be used to advantage in senile hemangiomas (ruby points) and spider nevi. If the spotty or branched telangiectases are a little more extensive, the microburner is unsatisfactory because even if care fully used, it often leaves an area studded with spots and scar pits. Therefore the method should never be used for the treatment of nevi flammei. In lupus vulgaris, which causes scar formation anyway the small scar pits from the microburner are not of great importance. Therefore it is useful for the removal of solitary refractory lupus nodules.

For the removal of the just-mentioned small tumors, telangiectases, and lupus nodules, *electrolysis* or needle *diathermy* may be used instead of the micro-burner However both methods also leave similarly pitted scars if employed effectively Furthermore electrolysis needs much more time. Both methods have also been recommended for the treatment of warts. Electrolysis and needle diathermy are also used *for permanent epilation* in hypertrichosis. In the treat ment of this condition the needle must be introduced alongside the hair into every single follicle Then a weak current is turned on for a certain length of time Frequently the hair root may be missed, and a new hair will grow after awhile, necessitating repetition. Of course for such a tedious method, only cases of hypertrichosis with so to speak countable hairs are suitable. If the hairs are too numerous or too fine, they cannot be removed in this fashion but must be sha ed or bleached and thinned with hydrogen peroxide and then rubbed off with pumice stone.

Epilations must be done very carefully and expertly since they may also leave small scar pits and even more extensive and very disfiguring scars which may interfere with facial mobility However it is probably not true that elec

the rotating grinder—and even more readily if this is a wire brush This may cause loss of control of the instrument and so produce lacerations and may also break the fast rotating flexible shaft of the grinder A field of not more than 2 × 2 cm is worked upon at a time An assistant surrounds the field with three thick gauze pads and sprays it with Freon for no longer than 20 25 seconds Then he quickly takes the protective gauze away and immediately the operator grinds off the board stiff purple surface with motions at right angles to the plane of rotation of the grinder usually in several crosshatched directions. This has to be done within a fraction of a minute before thawing occurs. Field after field is abraded until all the stain is removed With experience this operation takes less than 1 hour for a whole face The abraded face is dressed with non-sticking gauze (Telfa pads) and the patient is sent home To avoid edema he is advised either to sleep the first night after the operation sitting up in an armchair or with the head well elevated Exuding serum may be dabbed with sterile gauze pads until crusts form These are shed after about 1 week The healed skin is at first red and sometimes a little puffy but these after effects of the freezing or mechanical injury disappear within a few weeks.

Such abrasion or surgical planing may be repeated The operation requires experience and a technique which can be acquired only by assisting by working on anesthetized dogs, and to some extent by removal of seborrheic keratoses on the back where a little scarring is not of such importance as on the face of a young girl. Fresh pigskin stretched over a plaster cast of a face is suitable as a sort of manikin The main points of the technique are a firm grip and constant moving of the hand piece at right angles to the direction of rotation abrading thin layers successively without penetrating deeper into the corium than the papillary layer avoidance of sharp outlines or grooves avoidance of overfreezing The regeneration of the abraded epidermis takes place largely from the appendages of the skin which explains the rapid healing If skin is abraded so deeply that the appendages are destroyed an ordinary defect with resulting scar results. Therefore only skin which still has appendages is suitable for planing Scars which are too deep so-called "ice-pick" scars in some acne cases or appendage less residues of cystic acne should not be abraded because this operation would merely replace a scar with another scar

There are several types of machines on the market which differ in construction of the shaft and rotating speed With high velocity machines of more than 30,000 r p.m abrading can be done without refrigeration under procaine anesthesia but this method has not become popular in America because the dry frozen field is cleaner than the infiltrated field which is immediately covered with bloody ground tissue *The actual abrading tool is either a rotating* heatless carborundum or diamond stone a serrated steel wheel or a wire brush The wire brushes which are not only difficult to control but also hard to clean and expensive because of their short lifetime are now little used in America

a puncture in the nape of the neck instead of the lumbar area. It is indispensable in the treatment of syphilis because expert treatment of syphilis is impossible without examination of the spinal fluid. In recent infections this test is necessary for the establishment of cure, and in old infections regularly repeated checks of the spinal fluid often guide the entire treatment. Lumbar puncture is not quite satisfactory for obtaining spinal fluid because no matter which technique is followed the occasional meningism of several days which follows it may be so agonizing that it scares the patients away. Cisternal puncture, however, is not more of an operation subjectively for the patient than the puncture of a vein and even less, since the needle stab through the skin in the nape of the neck is usually felt less than in the flexure of the elbow. Substitution of lumbar puncture by cisternal puncture has a significance in the management of syphilis similar to the substitution of bismuth for mercury. Really, the essential success of the bismuth compounds does not lie in the fact that they heal syphilis better and are a little less toxic but much more because since their advent, the patient no longer runs away before termination of the treatment which was frequently the case when he had to stand the painful mercury injections. Therefore well-managed syphilis cures became more commonly possible only after the introduction of bismuth. By the same token, the general management of a more expert treatment of old syphilis infections has become possible because our patients are no longer scared away by the fear of pains which follow a considerable percentage of spinal punctures.

For a good cisternal puncture, a special needle and a suitable support for the patient's head are required, since the operation is best done with the patient lying on his right side. If cautiously and expertly executed, the operation is just as harmless as the spinal puncture. The right place for the puncture is simply the deepest point of the pit which is palpated above the vertebra prominens. If one encounters a bone, it can only be the occiput or the atlas. If the latter is the case and the needle is pushed farther downward, one is likely to get into a wrong direction below the atlas. Therefore, on feeling a bony resistance, one should always first feel with the needle in an *upward* direction. But the most important precaution is to stop the operation without hesitation if one does not succeed immediately and to try again a few days later. Then one usually succeeds without trouble. Such a repetition is possible with the cisternal puncture because the patient knows that he has to expect only a simple needle prick and therefore he will certainly come back if he cares at all for his health. Of course, it is not necessary that every dermatologist master the technique of cisternal puncture, but he who does not do so might profitably contact a colleague, either a dermatologist, neurologist or surgeon, to whom he can refer his patients for such puncture. This is not humiliating, since he can treat old syphilitic infections expertly only together with other specialists, such as ophthalmologists and internists.

trolysis stimulates hair growth in the neighboring follicles, as is often said. This impression has probably been caused by the fact that usually patients whose *hypertrichosis is still progressive* desire treatment. It is necessary to consider this at the start and inform the patient about the prognosis. Such surgical diathermy not only is a method for the removal of small tumors and hair but can also be used for performing larger operations. By employing an adjustable *wire loop* in place of the needle, the skin can be removed layer by layer without capillary bleeding. The electric loop can be drawn through the skin as if it were butter. Bleeding from larger skin vessels can be stopped by more vigorous coagulation after hemostasis is achieved with a metal pressure ring. Because of the absence or presence of bleeding, this method always permits us to know the layer of skin into which the loop cuts. The instantaneous stopping of capillary hemorrhage is a great advantage. There is no afterpain and the wound closes within a short time under a paste dressing, usually leaving a smooth, soft, and slightly depressed scar. Occasionally the scar may be hypertrophic. The method is most suitable for the removal of isolated small lupus patches on covered areas of the body (Fig. 140, p. 88) and on the ears, superficial epitheliomas, *rhinophyma, slow healing ulcers, and occasionally also tattoos. For good re*sults, a special apparatus is required.

In this connection it may be mentioned that some small tumors can very suitably be removed by chemical *caustics*. This, however, does not apply to ordinary warts, because keloids may follow, which, because of their unfavorable prognosis, are much worse than the original disease. This is true even though caustic methods are a favorite among the laity. Silver nitrate and fuming nitric acid are particularly dangerous. On the other hand, liquefied phenol acts too superficially. With tri- and bichloracetic acid, seborrheic warts and small xanthomas can usually be removed without the danger of scar formation. Small condylomata acuminata may be caused to dry and drop off by caustic powders (resorcinol, summitates sabinae, resin of podophyllum) or caustic tinctures, such as 20 per cent resin of podophyllum in alcohol. The latter application should, however, be used with the greatest caution on the vulva, under the prepuce, and around the anus, where moist surfaces touch each other, since severe inflammations have been observed. In a wider sense, dry ice and liquid nitrogen treatment, which has been discussed (pp. 270–71), may be classified with the caustic methods (cryocautery). Tattoos can be removed by denuding the skin with the microburner and rubbing potassium permanganate powder into the burn. The resulting black eschar will take the ink particles along when it is shed, but the remaining scars are often rather conspicuous. Finally, an operation must be mentioned which consists essentially of a stab of a needle only, but it is important enough in dermatological practice to deserve a special discussion, namely, *cisternal puncture*.

Cisternal puncture has the purpose of obtaining cerebrospinal fluid through

more quietly and accurately Even treatment with the microburner electrolysis (except epilation) and diathermy make local anesthesia with novocaine desirable in most cases. It has already been mentioned that in some cases (telangiectases) it is advisable to treat without anesthesia. On the mucosa, simple painting with 2 per cent butyn solution is sufficient. Of course it does not make sense to inject novocaine if the operation itself consists of a needle prick only Curettage usually requires only ethyl chloride spray for anesthesia. Only in exceptional cases, such as very extensive warts and condylomata acuminata may a *general anesthesia* with ether or thiopental sodium injection become necessary in dermatologic practice. Finally a few words on *prostheses* Syphilis and particularly tuberculous lupus may cause such horrible mutilations of the nose that the patient is rendered unfit for most vocations. These cosmetic invalids can still be helped by elastic artificial noses made of a mixture of glycerin gelatin and powder These artificial noses are almost indistinguishable from normal skin, especially since they move with the facial movements. Therefore, a dermatologic clinic should be concerned with the fashioning of such prostheses. Sometimes they are also desired for *traumatic* facial defects and for missing ears The elastic artificial noses have the disadvantage that they have to be freshly cast every day by the patient but most patients are glad to undertake this chore because to them it means return of the badge of membership in human society

The *injection treatment of varicose veins* also belongs in the domain of the dermatologists who by the way invented and introduced the method though it is now also done by many surgeons gynecologists and general practitioners. This procedure also requires special knowledge and training The injection treatment of varicose veins has the purpose by injection of an irritant of causing a localized inflammation and thrombus which slowly organizes and obliterates the diseased vessel At first it was a matter of concern that pieces of the thrombus might come loose and cause dangerous embolism but this did not happen The patient can go back to work after the injection and even do strenuous work unless a more violent and painful inflammation associated with the acute thrombosis incapacitates him for a few days The dangers of the injection treatment do not originate in the possibility of an embolism but rather in unsatisfactory technique Since the agents used are always irritating liquids even a few drops injected paravenously may cause necrosis and ulcers which may take months to heal This may happen even if the injection has gone off perfectly well but a few drops of the liquid seeped out through the puncture wound into the tissue Therefore the treatment should be practiced *only by physicians who have acquired great skill in intravenous injections* Immediately after the injection the vein must be compressed It is urgently recommended that the newer more innocuous agents be used e g sugar and especially salts of fatty acids such as sodium morrhuate. The sodium morrhuate preparations may rarely cause severe and even dangerous allergic reactions in persons allergic to fish proteins Before treating the patient one should inquire whether such an allergy exists, and a skin test or at least a preliminary injection with a tiny amount should precede the effective injections. Adrenalin solution should be in readiness for allergic shock The fact that months and years after treatment recurrent or new varicose veins not infrequently form does not diminish substantially the value of the method since the same also happens after surgical removal of the varicose veins, and such recurrences are often amenable to repeated treatment. Because of the danger *of paravenous injections the method should be applied only on relatively* strict indications An undoubted indication exists only if the patient has substantial complaints such as pain and fatigue in the leg or eczema or varicose ulcer Of course in many cases such as young persons female physical education teachers, and actresses purely cosmetic indications cannot be entirely ignored However even then one should be reluctant because the disfiguring scars which appear after faulty injections are still uglier than the varicosities and because even successful obliterations frequently leave pigmented streaks Of course the dermatologist must avail himself of *anesthesia* whenever it can be of help to the patient This needs to be emphasized because it is often disregarded owing to the smallness of many dermatologic operations Cosmetic operations under anesthesia also have better results, since they can be done

cillin in syphilis sulfonamides, penicillin, and other antibiotics in pyogenic and other infections mercury and arsenic in warts. Some dermatoses not of infectious nature or not yet so recognized may also sometimes respond specifically to certain medicaments lupus erythematosus to bismuth, gold quinine, and other antimalarial drugs, such as atabrine and chloroquine, lichen planus to bismuth arsenic, and Bellergal psoriasis and Boeck s sarcoid to arsenic pemphigus to ACTH corticosteroids, and Germanin dermatitis herpetiformis Duhring to sulfapyridine and sulfones arsenic and gold dermatitis to BAL and urticaria to antihistamines. Of course, when treating with these agents, one has to calculate the risk of toxicity Wherever two remedies are available, one being more effective but also more toxic than the other it is advisable to start with the less effective one. Thus the gold treatment of lupus erythematosus should not be undertaken unless the antimalarials, such as Atabrine, Chloroquine, Plaquenil or just quinine and even bismuth have been tried. Fortunately the antimalarials are more effective than gold which occasionally produces a most dangerous type of dermatitis and also melanoses. In this way the patient for whom the antimalarials or bismuth are sufficient is spared the dangers of gold therapy while the others have only the disadvantage of a delay which is not important in an extremely chronic disease It is also wrong to use very powerful agents in relatively harmless diseases which can also be treated with non dangerous methods. In this respect, arsphenamine should be mentioned. Arsphenamine an agent which had been a blessing to uncounted millions of sufferers from syphilis, has time and again caused the most severe toxicodermas and occasionally deaths, even though the usual and most well-tolerated doses were not exceeded Therefore, the treatment of psoriasis, tonsillitis, warts, etc. with injections of arsphenamine seems generally not permissible. Besides, there is little reason to expect that diseases which mostly or frequently respond to arsenic must also be expected to respond to arsphenamine. Granted, arsphenamine is an arsenical but the healing effects of arsphenamine, mapharsen, and related agents are highly specific, so that diseases which are cured by arsphenamine (syphilis) are usually not affected by inorganic arsenicals and vice versa and diseases which respond to the inorganic arsenicals (lichen planus, psoriasis verrucae) are mostly not influenced by arsphenamine If it is a question of using internal treatment or external treatment, one should not consider internal treatment preferable though the patient naturally will like it better because it is more convenient. Psoriasis, for instance, can be influenced internally especially by arsenic, as well as by ointments. Yet it is not right to substitute arsenic medication for treatment with ointments. Arsenic, aside from its hazards, favorably influences only a minority of the cases—at best, one third—and even these cases relapse, almost without exception after a few months. By contrast, a correctly executed external treatment heals almost every case of psoriasis, except the erythrodermatic ones. Recur

CHAPTER THIRTEEN

Systemic Treatment of Skin Diseases

OF COURSE there are also internal remedies in dermatology They may be administered orally or by injection and they may be symptomatic as well as specific. Among the *symptomatic remedies* the *antipruritics* are of major importance, pruritus being by far the most important subjective symptom. Mainly calcium injections bromides and various sedatives have been used to alleviate itching Hypnotics are also indispensable because itching most frequently occurs at night and long-continued insomnia caused by the itching may contribute to weakening the patient In the presence of severe pain one may resort to morphine but one should keep in mind that morphine is no antipruritic. Itching is sometimes more reliably relieved by the modern antihistaminic agents than by the older remedies mentioned Of course one should not omit the simultaneous use of external antipruritics such as menthol phenol resorcinol tar and cold compresses if the internal medicaments prove insufficient.

Violent scratching in general is not favorable for the healing of skin diseases. Eczemas may be aggravated and infectious dermatoses may be disseminated by scratch inoculation Ointments which have been applied are rubbed off and especially in children wounds capable of becoming new portals for infection are made. Therefore it is customary to tie the hands of small children to the bed frames or to splint their arms with cardboard cuffs so that they cannot reach their faces Corresponding measures in adults, like sealed dressings etc. are partly not practical partly useless, and in general not necessary Certainly the unfavorable influence of scratching is not sufficient to counteract good external treatment provided that care is taken that the rubbed-off ointment is soon replaced There usually is little point in prohibiting scratching when the patients already are so upset and worried that they cannot withstand the itching or that they scratch unconsciously while asleep It is the task of the physician to cure the itching Then the *scratching* will take care of itself Fortunately except in rare cases this can usually be accomplished in a short time by external methods of treatment including X ray

For some infectious skin diseases we possess *specific remedies* of varying effectiveness, such as sulfones in leprosy arsphenamines bismuth and peni

cillin in syphilis sulfonamides, penicillin and other antibiotics in pyogenic and other infections mercury and arsenic in warts. Some dermatoses not of infectious nature or not yet so recognized may also sometimes respond specifically to certain medicaments lupus erythematosus to bismuth, gold, quinine and other antimalarial drugs, such as atabrine and chloroquine lichen planus to bismuth, arsenic, and Bellergal psoriasis and Boeck's sarcoid to arsenic pemphigus to ACTH corticosteroids, and Germanin dermatitis herpetiformis Duhring to sulfapyridine and sulfones arsenic and gold dermatitis to BAL and urticaria to antihistamines. Of course when treating with these agents, one has to calculate the risk of toxicity Wherever two remedies are available, one being more effective but also more toxic than the other it is advisable to start with the less effective one. Thus the gold treatment of lupus erythematosus should not be undertaken unless the antimalarials, such as Atabrine, Chloroquine, Plaquenil or just quinine, and even bismuth have been tried. Fortunately the antimalarials are more effective than gold which occasionally produces a most dangerous type of dermatitis and also melanoses. In this way the patient for whom the antimalarials or bismuth are sufficient is spared the dangers of gold therapy while the others have only the disadvantage of a delay which is not important in an extremely chronic disease. It is also wrong to use very powerful agents in relatively harmless diseases which can also be treated with non dangerous methods. In this respect arsphenamine should be mentioned. Arsphenamine, an agent which had been a blessing to uncounted millions of sufferers from syphilis, has time and again caused the most severe toxicodermas and occasionally deaths, even though the usual and most well-tolerated doses were not exceeded Therefore the treatment of psoriasis tonsillitis, warts, etc. with injections of arsphenamine seems generally not permissible. Besides, there is little reason to expect that diseases which mostly or frequently respond to arsenic must also be expected to respond to arsphenamine Granted, arsphenamine is an arsenical, but the healing effects of arsphenamine, mapharsen and related agents are highly specific, so that diseases which are cured by arsphenamine (syphilis) are usually not affected by inorganic arsenicals and vice versa, and diseases which respond to the inorganic arsenicals (lichen planus, psoriasis, verrucae) are mostly not influenced by arsphenamine If it is a question of using internal treatment or external treatment one should not consider internal treatment preferable though the patient naturally will like it better because it is more convenient. Psoriasis for instance can be influenced internally especially by arsenic as well as by ointments. Yet it is not right to substitute arsenic medication for treatment with ointments. Arsenic aside from its hazards, favorably influences only a minority of the cases—at best, one-third—and even these cases relapse almost without exception, after a few months. By contrast a correctly executed external treatment heals almost every case of psoriasis, except the erythrodermatic ones. Recur

rences can usually be cleared or checked again because one has had the opportunity to become thoroughly familiar with the specific healing reactions of the patient while treating the eruption. Arsenical treatment without simultaneous external management *therefore means a waste of time for almost all psoriatic patients.*

While formerly the *vitamins* were used only to balance the deficiency of vitamins in the relatively rare avitaminoses such as pellagra and scurvy they have more recently been used in fantastically high doses as *specific drugs*. Much of this knowledge is still in the experimental stage with more *optimism than success*. The endless experiments in this direction are very understandable after *we have learned that with the help of vitamin D_2* one could cure tuberculous lupus by daily swallowing a couple of tablets; the treatment of this disease previously necessitated a special department with expensive equipment and specially trained staff in each skin clinic and imposed decades of torture on the patient with painful applications of caustic ointments and endless radiations. It is a remarkable fact that the same vitamin has but a very *minor* effect on the other forms of skin tuberculosis and also on the tuberculids. On the other hand it has also seemed to influence favorably some skin diseases which have nothing to do with tuberculosis such as acrosclerosis and chilblains. More recently the less toxic and more rapidly effective isonicotinic acid hydrazide (Isoniazide) has almost replaced vitamin D_2 in the treatment of lupus vulgaris. Para-aminosalicylic acid and streptomycin are also useful antituberculous drugs.

The specific remedies also include the vaccines. In dermatology tuberculin has been used in the treatment of tuberculids, trichophytin in deep trichophytosis and staphylococcic vaccine in acne and furunculosis. In venereology gonococcic vaccine has applications in gonorrhea and Dmelcos in ulcus molle. The role of vaccines is however not an important one. In stubborn skin infections, especially in staphylodermas and streptodermas, autovaccines are made, and some observers feel that they are endowed with a special healing power. *In recurrent herpes simplex* the recurrences can sometimes be prevented by repeated smallpox vaccinations. Prior to the advent of the antibiotics the dermatologist had *very few internal remedies which deserved the attribute specific*. Penicillin at first regarded as remarkably free of side effects, gave us specific help in pyogenic infections and syphilis. Soon however its splendid reputation was marred by the frequent allergic reactions, especially stubborn urticaria which its internal administration caused. But the worst was still to *come*. At the present time a number of fatalities due to acute anaphylactic shock within minutes after the injection of penicillin have been reported and more have probably occurred. Oral administration is also dangerous and in extreme cases, even the scratch test may cause dangerous reactions.[1] The

1 Such cases being very rare scratch tests with penicillin before penicillin therapy have been *recommended* by allergists. If the scratch elicits an immediat rticarial reaction, penicillin should not be given. Whether a negative scratch test rules out existing llergy t penicillin *is not so certain.*

newer antibiotics (Aureomycin Terramycin and other tetracyclines) have so far not shown these dangerous allergic side effects, so that their use is safer though more expensive The development of pruritus ani and moniliasis after the use of the tetracyclines is an annoying though not dangerous aftereffect. No penicillin injection should be given without having adrenalin (epinephrine) in aqueous solution ready for intravenous or intramuscular injection. The *adrenal cortical steroids* (ACTH, cortisone etc) are effective specifics in pemphigus and systemic lupus erythematosus (see paragraph on hormones, p 296)

If the effect of the vaccines which are considered highly specific is not very distinct and even doubtful, this is still more true of the *non-specific agents* which are in use in dermatology In spite of often liberal recommendations and wide acceptance a clear-cut healing effect can be observed only in some special cases.

First, there are certain *intravascular agents* (calcium and strontium gluconate injections) In very acute eczemas with severe pruritus and in other cases of pruritus, they sometimes seem to exert a soothing influence and in certain cases of urticaria the effect is immediate and without doubt. However their use in chronic skin diseases and especially in chronic eczemas seems to lack empirical foundation.

The classic drug for the non-specific treatment of all kinds of skin diseases was *arsenic* Its undeniable effect on lichen planus and some cases of Boeck's sarcoid, psoriasis, and verrucae has already been mentioned. Some cases of tuberculids, pemphigus, and mycosis fungoides (granuloma fungoides) also respond favorably Its success in certain blood diseases, including some which cause skin manifestations such as leukemia and hypochromic anemia, is also well known. However it seems unproved and unlikely to me that arsenic should influence favorably such heterogeneous skin diseases as eczema, acne alopecia, *etc* In part this belief seems based simply on errors of observation. For that matter I am inclined to think that the esteem in which arsenic is held in the treatment of chronic eczemas and especially of its chronic papular and licheni fied forms is due to occasional confusions with lichen planus. The custom of trying arsenic if one is at one's wit's ends with a refractory skin case should be given up the more so because the dermatologist can expect a clear effect from this agent only if *strong* treatment is applied even frequently exceeding the maximal dose, e.g also the maximal dose for the single injections in a series. But *strong* arsenical cures cost much time and patience and in most patients lead to transient symptoms of poisoning This, at least, is true of its oral ad ministration while parenteral administration has the great shortcoming that the patient *must* visit the physician daily The toxic symptoms, though not dangerous in treatments shorter than 6 months, are still often quite trouble-some Most frequently observed are lassitude, depression stomach-ache and diarrhea Loss of weight is the rule in such series, in contrast to the weak arsenical treatment given on the grounds of internal indications, such as gen-

eral debility and anorexia This type of administration is more likely to cause a gain in weight Besides the gastrointestinal mucosa other mucosae may react with inflammations Conjunctivitis bronchitis, and urethritis are examples. Arsenical erythemas may be painful on the palms and soles. On the trunk, the large flexures, and pressure points they may vanish very slowly leaving leukomelanodermas For all these reasons the patient needs continual weekly checks by the physician

If arsenic is given orally tolerance develops rapidly probably because less and less arsenic is absorbed by the gastrointestinal mucosa. Owing to this tolerance the drug gradually agrees better with the patient, but in the same measure it also loses its efficacy *Therefore it becomes necessary to increase the dosage regularly and systematically* For this reason one starts with a few drops and increases the number of drops by one drop daily or every few days. If symptoms occur the number of drops is not increased When they subside the dose is increased again If the complaints are very marked it is advisable to return temporarily to half the number of drops and then start increasing again If the series is over an old ritual demands that one slowly reduce the number of drops. This is useless and a waste of time since there are no symptoms of abstinence. It is better simply to discontinue the treatment The administration of pills which are better tolerated because they are more poorly absorbed has no advantage in comparison with drops

It is not to be assumed that arsenic influences skin diseases in weak or even homeopathic doses. Therefore such types of administration are not justified This does not mean that *homeopathic doses of certain drugs are never justified* Very small doses of certain medicaments are successfully used not only in internal medicine (iodine) but also in dermatology (e g gold in lupus erythematosus) Some authors like to explain this fact by a catalytic effect of the agents. No sensible orthodox physician will be opposed to the administration of homeopathic doses if their effect is made probable on grounds of experience, and the dermatologist has the least reason to do so In external treatment homeopathic doses are often used in trying to overcome sensitivities of the skin In such cases e g with chrysarobin one starts treatment with doses of 0 1 per cent or even less and not rarely succeeds in accustoming the skin to the drug so that one may finally use the drug vigorously In the case of tolerance to allergens which have caused eczemas before especially occupational eczemas the rule of homeopathy that the disease should be treated with the same agent which has caused *it is also followed* The principal difference between homeopathy and orthodox medicine is not that one school of thought treats with small doses and adheres *to the principle of similia similibus curantur*[2] while the

2. Latin, literally "to heal like with like." This is the principal axiom of homeopathy namely t heal a disease with an agent which if taken in toxic dose produces symptoms simila to *those of the* disease

other does not, but rather in the fact that the homeopaths have lower standards for accepting empirical proof of therapeutic effect. So-called orthodox medicine has developed and become a blessing to mankind because it has studied the pitfalls of empirical observation and has largely succeeded in eliminating them. Such pitfalls are the false logic of *post ergo propter* (Latin "after therefore because") the error of too small series, the unconscious preselection of cases, the lack of a clear-cut and lucid arrangement of experiments, and the unreliable testimony of witnesses. Orthodox medicine will adopt any method of treatment which passes the sound test of empirical value, even the most lowly one. It certainly is not true that scientific medicine shuts itself off against homeopathy but it is true that scientific medicine refuses to embrace "homeopathism" which is the state of mind which in the process of appraising the effect of healing measures, declares itself satisfied with theoretical considerations and occasional or scientifically uncontrolled impressions. On the contrary scientific medicine relies on the truthful testimony of an experience which has been checked with stern and critical skepticism and discards every principle of treatment which fails to pass the empirical test. What has been said about homeopathy applies, of course, to other unorthodox methods.

Still more than with arsenic, we become involved in theoretical speculations and uncertain observations if we turn to those remedies which are supposed to heal skin diseases by *Umstimmung* which, in German literally means "change of tune" but may be translated as "alteration" meaning a mysterious change of performance in the system. Other such ill-defined conceptions are *detoxification* and *mobilization of the defenses* Granted, there exist some such medicaments, the application of which in well-defined diseases is sufficiently well founded on experience There is no doubt that urticaria is frequently cured by *alteration* caused by a single or a few injections of calcium gluconate, the patient's own blood, or adrenalin. *Detoxification* by antihistamines has also shown distinct and certain successes in urticaria However it appears to be an entirely different matter if we ask what these agents have accomplished in other diseases especially in those which have also been labeled "allergic" and which play the most important role in dermatologic practice namely the acute and chronic eczemas. Here the situation seems to be similar to the application of heat which works well with certain more deep-seated dermatoses, such as boils and deep trichophytosis, but fails us when we need it most (eczema psoriasis, lichen planus) or even tends to make things worse Thus the mobilization of the biological defenses by heat or by provocation of an erythema (alopecia areata) is valuable only in certain cases and has no generally applicable indications

These examples show that there is justification in saying that such agents have frequently been accepted with too much faith. There are dermatologists who whenever there does not seem to be sufficient progress, give injections of

turpentine or of the patient s own blood Others have other favorite medications—for example injections of milk which however should be handled with greater caution because of occasional though rare anaphylactic incidents. Injections of sulfur oil and fever therapy are less popular because of the accompanying pains and other inconveniences. The popularity of this kind of therapy is decided by particular vogues which may last for longer or shorter times and also by the efficient advertising of the pharmaceutical industry The physician should always keep in mind that he should trust a medicament the less the larger the list of its indications and the more general and ill defined the conception of its effectiveness.

Among the drugs with much too general indications are also the *hormonal preparations* It is really remarkable how little of the high expectations which accompanied their appearance in the field of skin diseases has materialized except for corticotropin and the adrenal cortical steroids. Of course one should free one s self of the idea that a disease must respond to hormones because it takes place in an organ which is subject to hormonal influence as, for instance with regard to hair By far the majority of cases of hypertrichosis and alopecia are caused by local factors and have nothing to do with endocrine abnormalities In these cases hormonal treatment would simply be erroneous The most vexing thing however is the fact that even in hair and pigmentary anomalies whose endocrine pathogenesis seems to be obvious no clear-cut successes have so far been obtained In fact in some cases the condition seemed to get worse At any rate there is absolutely no justification for treating with hormones such dermatoses as vitiligo about which not only have no successes become known but whose connection with endocrine secretion remains completely hypothetical However there is a remarkable exception to the relatively small value of the hormones in dermatology During the last decade a most valuable series of hormones has become available namely cortisone and some newer derivatives such as hydrocortone and the pituitary adrenocorticotropic hormone (ACTH) which activates the adrenal cortex All these corticosteroids have in common that they stop most inflammations, especially the acute forms provided that they are given in sufficient dosage For the first time we have at our disposal a reliable internal treatment of acute widespread contact or drug dermatitis and of acute episodes in the course of atopic eczema and urticaria These hormones often give the patient quick relief *from itching* pain oozing and other discomfort particularly and most economically in the form of ACTH given by slow intravenous infusion (*20 30 mg of ACTH in* 500 cc. of normal saline 30 drops per minute) This is a hospital procedure but these hormones can be given by *intramuscular injection* and the corticosteroids preferably by the oral route Unfortunately the sometimes miraculous effect achieved does not last much longer than the application of the drugs but in such self limited diseases as acute contact dermatitis or some forms of urticaria

or drug eruptions the patient may well be tided over his disease by these essentially symptomatic and not disease-specific drugs. In the two fatal skin diseases, namely pemphigus and acute systemic lupus erythematosus, they are of the greatest value. Since it is possible to administer them with care over many years without great damage, a patient with pemphigus can be kept symptom-free by sustained medication much as a diabetic is kept alive with insulin. However with regard to many other hormones, hormonal therapy has too often been based on theoretical expectations rather than on empirically ascertained successes.

The conception of healing the system by *detoxification* has led to the same kind of wishful indications. Of course it is an understandable desire, especially in the case of toxicodermas, to bind the poison in the system or to eliminate it rapidly. For decades, this has been the reason for administering sodium thiosulfate in gold arsphenamine and other forms of drug dermatitis, and there also was a good theoretical foundation for doing so. However the actual performance has not passed the test of critical observation. With the advent of BAL, we at last have been given a medicament with undoubted detoxifying properties. It has, however a very specific indication for certain metal poisonings (arsenic, gold) and it certainly is unable to eliminate all kinds of "poisons" from the system. Nor can the skeptical investigator say that the laxatives which in the layman's opinion are always indicated and the supposed intestinal disinfectants (sulfur ichthyol) have any convincing effect on skin diseases, again with the exception of well-known specific instances, such as urticaria. But therapy takes on a truly medieval character if the dermatologist accepts the viewpoint of many patients who just in skin diseases desire something to "purify the blood" Such requests are quite regularly voiced during the treatment of eczemas, psoriasis, acne and chronic pyodermas. Obviously the old conceptions of acid in the system the old "acrimonia sanguinis," are still alive in spite of the fact that these hypotheses have done nothing for practical therapy but have stymied the progress of specific and empirical therapy for centuries. Therefore, the serious physician must honestly ask himself whether he should yield to the delusion of the patient in order not to lose his confidence or whether he should explain the truth to him in order to keep him more securely attached to a therapy whose chance is based on experience and thus to induce him to endure more patiently the inconveniences of the external treatment. In general it certainly will be better to choose the way of instruction and enlightenment if for no other reason than because it is the honest one. With uneducated and unintelligent persons, however it can be doubted whether the way of honesty will lead to the intended goal and, therefore whether it is really the most suitable method to help the patient.

The desire for "purification of the blood" coincides with the desire to treat the metabolism and the *constitution* or as it is more recently called the

turpentine or of the patient s own blood Others have other favorite medications—for example injections of milk which however should be handled with greater caution because of occasional though rare, anaphylactic incidents. Injections of sulfur oil and fever therapy are less popular because of the accompanying pains and other inconveniences. The popularity of this kind of therapy is decided by particular vogues which may last for longer or shorter times and also by the efficient advertising of the pharmaceutical industry The physician should always keep in mind that he should trust a medicament the less, the larger the list of its indications and the more general and ill defined the conception of its effectiveness.

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in dermatoses but even in syphilis. It is true that there are certain skin diseases which rapidly vanish in a spa or even after mere hospitalization without other treatment (prurigo certain cases of atopic dermatitis, strophulus) It is widely assumed that these may be caused by allergens or other factors in the house. But it is not permissible to send such patients to expensive resorts, because the eruption almost always recurs rapidly when the patient returns to his old surroundings. Naturally in other cases of stubborn skin diseases climatic changes and recreational treatment are still less in order The patient with a chronic dermatosis does not belong in a spa but in a dermatologic hospital where he can be systematically treated by trained specialists.

Incidentally the regular *physical activity* which is regarded as an important factor at spas is undesirable in the treatment of most chronic skin diseases, since heat and friction are likely to increase inflammation Here, too it is true that what is good for the healthy and for many internal patients is mostly wrong for the skin patient Also for example, warm and especially woolen clothing which the sick and convalescents generally need, is frequently not well tolerated by skin sufferers.

There are some special reasons for the *unjustified esteem* in which the public holds general supportive measures with regard to the treatment of skin diseases and the physician should know them. In the first place there are some skin diseases which can be influenced by *suggestion*. This certainly is true of warts and granuloma annulare and possibly also of other diseases (urticaria and psoriasis) This may cause the false impression that one of the general measures has a specific effect. Second, as has been mentioned some skin diseases apparently are caused by *home allergens* and therefore will be benefited by climatic cures Such an improvement is likely to be credited to the improvement of the general condition of the patient. In the third place quite a few skin diseases (eczemas, psoriasis, strophulus furunculosis, pemphigus, mycosis fungoides) exhibit striking *fluctuations of intensity* For unknown reasons, they may improve seasonally or vanish entirely or erupt and spread suddenly and then the puzzling change is likely to be explained by the treatment which had been given just previously And, finally in treating certain dermatoses, one gains the impression that they sometimes unexpectedly heal following a "push" of some kind but it is also true that they may get worse just as unexpectedly This is particularly true of psoriasis, and a few times I was impressed by such surprises in mycosis fungoides. The mechanism of these reactions is enigmatic, their laws and regulations still being unknown. Some of them might be credited to suggestion. At any rate it is curious how frequently psoriasis improves immediately following unusual events such as treatment with the most heterogeneous medicaments, change of locale psychic traumas, and intercurrent diseases. But duplication of such a sequence either in the same or in other patients usually fails. This phenomenon can be well exemplified from the literature,

entire system to elicit a general "mobilization of the defensive powers." This desire is time and again expressed by sufferers from eczema and psoriasis. Of course, in this case, too there are certain indications which the good physician will not miss. I mention as an example senile pruritus, which in most cases is a manifestation of diseases of other organs (dyspnea hypertrophy of the prostate) the treatment of which may also have a bearing on the skin disease. It is of course well known that the correction of metabolic diseases for example diabetes may favorably influence the course of accompanying skin diseases such as xanthomas furunculosis, oidiomycosis. But such special facts should not be sufficient reason for us constantly to pester all other patients with skin diseases with blood and metabolic tests and general restrictions. Even if one occasionally found diabetes in a psoriatic this would be just a coincidence, and there would be no reason to assume that the psoriasis would vanish following control of diabetes Nobody will assume that a syphilitic patient with psoriasis will get rid of his psoriasis if his syphilis is cured Even if the connection of a dermatosis with an internal ailment is definitely not coincidental as for instance in asthma and atopic dermatitis the healing of the internal disease by no means permits expectation of benefit for the dermatosis. There have even been claims to the contrary in this latter case namely that the eczema is likely to get worse for which there is no proof either There is also no basis for such general expectations as for instance, that a pale anemic child will necessarily lose his eczema, once his general condition improves. Whoever *treats a patient with a skin disease who is suffering at the same time from any* other troubles, internal or otherwise and leaves these ailments untreated or fails to arrange for a thorough examination is a poor physician But he *is not a* good physician either if he expects from such non-dermatological treatment a necessary improvement of the *skin disease* All that counts in the practical application of constitutional pathology is that the physician know *in which special cases* a relation of the dermatosis with the general condition of the system or *with other organs can be expected* and *that* he not hesitate to consult and co-operate in all these cases with the family physician or a suitable specialist sparing all other patients superfluous examinations and medications. *This alone* is true constitutional therapy The treatment of the "whole system" or "the individual as a whole" which has recently been postulated with so much noise thus turns out to be in one group of cases a matter of course which the good physician has never neglected anyway and in the rest an unnecessary burden resulting from the exaggerated generalization of individual cases. In our *practice* we deal almost exclusively with "*mere*" (*autonomous*) *skin diseases* in which improvement by *improvement of the general* condition of the patient or by healing of other diseased organs is not to be expected on the basis of experience. For this reason we have completely *abandoned the general climatic or spa treatments* of skin diseases which once were considered so important not only

in dermatoses but even in syphilis. It is true that there are certain skin diseases which rapidly vanish in a spa or even after mere hospitalization without other treatment (prurigo certain cases of atopic dermatitis, strophulus) It is widely assumed that these may be caused by allergens or other factors in the home. But it is not permissible to send such patients to expensive resorts, because the eruption almost always recurs rapidly when the patient returns to his old surroundings. Naturally in other cases of stubborn skin diseases, climatic changes and recreational treatment are still less in order The patient with a chronic dermatosis does not belong in a spa but in a dermatologic hospital where he can be systematically treated by trained specialists.

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which shows that almost every new method of treatment at first helps some psoriatics but soon loses its efficacy In psoriasis, one should guard one s self against haphazardly prescribing and trying whatever the prescription blank has room for particularly expensive or still worse, aggressive methods, the more so since we have at our disposal well tried and quite satisfactory remedies for this disease.

Diet treatment is another topic which deserves a place in the ill-defined chapter of constitutional therapy Here too there are a few special indications which however are mostly uncertain or inefficient. Probably best founded is the influence of a low fat or fat-free diet on some varieties of xanthoma and perhaps also in some cases of psoriasis. In practice however it does not help as much because the good execution of such a diet encounters infinite difficulties and actually necessitates hospitalization for months And besides this, on discontinuing the diet recurrences can be expected immediately For a time the success in tuberculosis luposa of the salt free diet which is easier to carry out than other diets aroused great enthusiasm but many of those who tried this method were disappointed

Of course diseases which are caused by *deficient and faulty nutrition* may develop *skin symptoms* (xanthosis by eating too much spinach and carrots pellagra and scurvy from specific vitamin deficiency) This however does not mean that special diets or vitamins are in order in the treatment of all sorts of dermatoses with entirely different etiologies Even vitamin D_2 which performed miracles in tuberculosis luposa failed to influence other forms of tuberculosis, let alone other diseases Apparently it is the same with the diets and vitamins as with the chemically specific drugs, for example arsphenamine. Though arsphenamine heals recent syphilis it is entirely ineffective in tuberculosis. It is not even effective in some late forms of syphilis such as general paresis tabes, atrophy of the optic nerve, or aneurysm of the aorta which latter it might even make worse

For the dermatologist the most important practical question about the diet problem is how the eczemas respond to this type of treatment Controversy about this topic goes way back in dermatologic history It was particularly spirited after a witty quip by the pediatrician Czerny Czerny opposing the dermatologist Neisser who pleaded for external treatment said 'Our colleague Neisser forgets that there is a child in the skin he treats. This witticism undoubtedly is convincing by suggestion The facts however speak much less clearly It is true that in pediatrics the conviction still prevails that *infantile and childhood eczemas* are caused by malnutrition though it cannot be stated what exactly is wrong with the diet Most frequently blamed are overfeeding and breast feeding which are the most common peculiarities of nutrition at these ages and therefore are likely to be most frequently suspected But the situation becomes still more uncertain as soon as one wants to know which diet

forms should be prescribed to heal these eczemas. Then it becomes apparent that there are no prescriptions which might resemble a law One pediatrician prefers one diet another pediatrician another one, and many have arrived at the basic principle that the main thing is to change the regimen under which the eczema developed, in other words, to prescribe a "contrast diet. It is diffi cult for the dermatologist to appraise the success which pediatricians have had with such diets. In any case, the great differences in the methods alone tend to show that the results are not very obvious. Many infantile eczemas heal well under external treatment, but extremely stubborn cases are no rarity either Therefore, it is advisable to try a diet treatment only with babies who fail to respond sufficiently to external treatment. In these cases the diet treatment should certainly not be delayed too long, so that anything that has been thought promising by competent observers may not remain undone.

The skeptical attitude of the dermatologist with regard to the dietary treat ment of infantile eczema can be partly explained by the historic development of dietary therapy of *adult eczema* There was a time when even scabies, which in a purely morphologic sense is a papulovesicular eczema, was considered a systemic disease and treated accordingly Since then the conviction of the use lessness of dietary regimens for adult eczematics has increasingly gained ground. Darier says aptly "There was a time when physicians believed in a nutritional regimen for sufferers from skin diseases and some still believe in it. But the congregation of the faithful is becoming smaller and smaller At any rate a rational diet for the ailing skin is unknown notwithstanding the exceptions we have already mentioned Above all, nobody has ever shown with cer tainty that eczemas of adults can be influenced favorably by general dietary regimens. Of course one should in this respect, recognize that intertriginous eczemas are more common and more difficult to treat in obese persons. But even in these cases one is still more likely to succeed with ointments than with the usually hopeless treatment of the obesity

The foregoing discussion should tend to make the dermatologist reluctant to be too liberal with dietary prescriptions in the treatment of skin diseases and not yield too easily to the suggestions and requests of the patient, because by doing so the physician will acquire unsound habits. Therefore, it is better to tell the patient frankly that experience has failed to show the value of dietary treatments. Insufficiently founded dietary treatments should also be refused because they are by no means innocuous methods. Especially in children one should be aware that they can easily impair the general nutritional condition of the patient The patient is served best by guiding his entire energy to the external treatment which promises most, provided that it is patiently executed. The patient is well served if his good intention to undergo external treatment is not weakened by methods which he may deem more important but which in reality are useless. Anything that may distract from regular and conscientious

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a thorough investigation for alimentary allergy One starts with an entirely bland *elimination diet* e.g only tea with zwieback or mashed potatoes, and adds one food item after the other Sometimes it is necessary to try to provoke an eruption with such foods as are suspected to be allergenic. In this way the search for an alimentary cause of the disease may be successful or one may at least arrive at the conclusion that the disease which gives the impression of an autointoxication and in some cases actually is cannot be shown to be a food allergy in a specific case. This at least frees the patient from the nuisance of useless diets. In the case of strophulus, we have freed patients in general, since it could be shown in tests which were repeated over and over again that provocation with the foods incriminated in all textbooks never caused new lesions. From these experiments, we concluded that strophulus is not caused by alimentary allergy and that dietary prescriptions are not indicated.

external treatment should be omitted in the patient's best interest. It is by no means necessary that a patient who wants to live as a vegetarian be explicitly forbidden to do so But the physician should tell him he is not interested in such a diet because it will not cure the eczema and that he is confident that he can heal it without such experiments

A word should be said about those skin diseases which are not caused by malnutrition but develop at normal nutrition because of a special alimentary hypersensitivity of the patient for example urticaria from sensitivity to oysters It goes without saying that in these cases dietary prescriptions though of a very special nature become essential It is necessary only to know which ones. In a given case this is often very difficult to ascertain because the method of injecting food-extract antigens under the skin to elicit an "immediate reaction" is unreliable If the alimentary hypersensitivity is directed against foods which are rarely eaten such as oysters and strawberries no physician is needed for the diagnosis, since the patient has actually already done the detective work himself But if the allergens are frequently used like eggs, cheese or fish the diagnosis is difficult In any case it is useless just to forbid something haphazardly It is particularly senseless if patients with urticaria stop eating strawberries and seafood because they have heard that other patients get sick from these items It is not without risk if the patient on erupting with new lesions entirely eliminates from his bill of fare all those food items which he had eaten shortly before because he believes them to be the villains. This situation is sometimes encountered in patients with strophulus and acne and may lead to a diet stripped of so many important articles that malnutrition develops.

In all these cases the dermatologist must start from the fact that in those diseases which empirically may be caused by food allergy (urticaria) in the overwhelming number of cases food allergy cannot be found and the search for an allergy is unnecessary if the eruption heals as it usually does after administration of tried remedies (calcium injections autohemotherapy with whole blood antihistamines etc) In these cases the search for an allergen would only mean a waste of time and unnecessary expense If however the case proves to be a resistant one there is no point in taking a long and unreliable history or to do dozens of skin tests which are still in the research stage and therefore do not permit a clear-cut answer but it is necessary to examine the diet thoroughly If one suspects some special foodstuffs such as eggs or cheese in acne and strophulus, an ambulatory treatment may be tried The plan must be discussed in detail with the patient and its execution must be checked regularly One advises the patient to eat eggs regularly for two weeks and to refrain from eggs for two weeks These alternating turns should be repeated as often as necessary There are however only very few patients who carry out such investigations conscientiously In more serious cases for example severe urticaria it is best to hospitalize the patient without delay and to start

but they are a handicap for practice. The therapist should not ask which healing mechanism is well conceived but he should concentrate his whole attention on establishing which remedy has, in the disease in point, according to experiences to date, exhibited the *greatest healing power* This is the remedy with which he must by all means start the treatment—of course, with due consideration of its toxicity and other disadvantages.

Frequently it is not a simple matter to find out which drug has the best chances in a certain disease. Naturally in most cases, our decision cannot be based on our own experience especially not in the rarer diseases, but must utilize the experiences of others as deposited in books and journal articles. In principle this is well possible. Nobody has ever seen with his own eyes that the earth rotates around the sun but rather only the opposite. And yet our conviction of the planet nature of the earth is not based on faith but on experience But, since most knowledge, including knowledge of healing is second hand knowledge it is necessary to scrutinize the offering hand.

The adequate critical appraisal of the material on the effect of drugs which is communicated to us by books and periodicals must be learned by everybody who wants to become a good physician. It is relatively safe to trust statements referring to drugs which for decades have been in use and have become standard material of the textbooks. Nevertheless, there exists the hidden danger that the application of a drug may have become a ritual which is passed on from generation to generation though its value has really never been accurately appraised. This occurs especially in dermatology where elaborately compounded prescriptions which somebody recommended a generation ago for reasons unknown are carried over from one textbook to the next. Many of these prescriptions have never been scrutinized for their healing value but are used because of favorable impressions. Occasionally these routines are entirely without justification, as has been demonstrated by the example of Dreuw's ointment which, in spite of its remaining very popular over a period of thirty years, is much inferior to an ordinary chrysarobin paste. Appraisal is still more difficult if one is dealing with *new* therapeutic methods as they appear and are recommended in the medical journals It is rare that effects are so obvious as in the cases of arsphenamine or sulfonamide In such a quandary the appraisal may be made easier by knowing how reliable the *author* is who recommends the remedy A wit once said that the most important benefit one derives from attending conventions is to find out those colleagues whose articles one does not have to read There is a great deal of truth in this cynicism. Personal acquaintance with the authors greatly facilitates the critical appraisal of their published experiences and saves much time for the physician who must keep abreast by reading. This is all to the advantage of his patients.

Considering the international character of our medical literature it can hardly be avoided that we have to rely in our therapy on the statements of com

CHAPTER FOURTEEN

Therapy and Experience

THE manifold temptations to therapeutic unsoundness which also face the dermatologist in the guise of so-called shotgun methods constitutional therapy and dietary regimens aided and abetted by unreasonable patients, make it necessary to take a closer look at the *purely empirical character of our therapy*

The effect of arsphenamine on syphilis is so clear cut that if one injects it into the arm one can with a stop watch observe the vanishing of spirochetes from the chancre. The thing runs off like clockwork. But arsphenamine is by no means directly toxic to the spirochetes. It may even be added to media used for culturing spirochetes. Nobody knows exactly why it affects syphilis so miraculously if it is injected into the bloodstream But however little we may know about it this lack of knowledge has nothing to do with the *practical value* of the drug arsphenamine which has been established empirically

Another example is the history of the discovery of germanin as a remedy for pemphigus which was prompted only by the vague idea that pemphigus the etiology of which is unknown could possibly be an infectious disease and that germanin might possibly help since it had helped in an entirely different but certainly infectious disease namely African trypanosomiasis. This surely is an insufficient argument but nevertheless it led to an important discovery It happens sometimes that somebody dreams up something which just does not make sense and yet may turn out to be a new method of healing Based on our knowledge it could have been expected that an artificial thrombosis of the veins of the legs would entail the danger of embolism and the surprise was great when this apparently dangerous experiment finally turned out to be harmless. In this manner the modern treatment of varicose veins was inaugurated Of course the opposite may also happen namely that an apparently permissible new operation proves to be most dangerous—even fatal—as was the case with the original thyroidectomy in which the parathyroids were also removed causing tetany

These cursory examples may serve to show that it does not matter at all whether one knows how the healing effect comes about Theories on the methods may have great scientific interest and be a great stimulant for research

suggestion of a resident to try to cause an alteration in the reaction of the skin by injection of specially prepared blood from the patient. But since I did not have enough suitable glass tubes on hand the injection could not be given on the same day and the patient was told to return two days later When he returned the eczema had vanished and the patient stayed well during many months of observation.

If on that day we had had at our disposal the necessary tubes, the consequences would have been incalculable. We certainly would have been duped by the obvious 'success and any doubts would have been dispelled by the optimism of the resident who originated the idea. In the time following the incident, we would have treated all stubborn eczemas with this method which had seemed so promising and it might have taken years until we finally became convinced that the method was ineffective Even then we might possibly have said based on our experience "And yet *once* I saw a patient get well with this treatment. Thus we can eliminate coincidence by enlarging our sample. The power of coincidence decreases in proportion to the increase in the number of cases. But the influence of coincidence depends not only on the size of the sample but also on the number of positive cases occurring in the sample. This is the basis for the calculation of chance which is the factor of uncertainty caused by coincidence inherent in a series of observations. This calculation may be computed according to the following formula

$$m = \pm \sqrt{\frac{p\,(100 - p)}{n}}\ \text{per cent}$$

This is called the ***probable error of the small number*** In this formula, n represents the total number of the examined individuals and p the number of individuals exhibiting a certain characteristic in the case of therapy improvement or cure.

The number obtained means that a statistical result which lies within the limits of this number may only very exceptionally be due to chance. For example, if I had treated 98 random cases of psoriasis with a new method and 19 cases (= 20 per cent) had been cured, the probable error due to the limited number would be

$$m = \pm \sqrt{\frac{20\,(100 - 20)}{98}}\ \text{per cent} = \pm \sqrt{16.2} = \pm 4\ \text{per cent}$$

The statisticians often triple this percentage in our case, ± 12 per cent. This would mean that the number of cases which have been cured by the new method lies between 8 and 32 per cent (20 ± 12 per cent) if there were no other factors influencing the result However in the treatment of diseases many known and unknown factors influence the outcome These factors do not enter the formula and therefore cannot influence its value However it is of value to be aware of

pletely unknown authors Then we must detect their reliability from the manner in which they describe their observations. We have to search for all those pitfalls which we ourselves should consider constantly in the formation of our *own* experience namely the error of small numbers the unconscious preselection of the confirming cases and the unclear arrangement of experiments.

The great enemy of all true research is the chance meaning the *coincidence* of two events without causal relationship It is logical that coincidence *is bound to happen* in a certain percentage of cases, with the consequence that the observer interprets the later event as an effect of the preceding event. This is the error of *post ergo propter* (after therefore because) One is inclined to believe that because a disease improved *after* the administration of a medicament it has improved *because* of the administration of the medicament. The sequence in time suggests a sequence of cause and effect. The danger of such misinterpretations is greatest if our observations are based on a single case or only a few cases. If we desire to learn how to appraise the healing effect of the medicaments we are using we must observe the effect of the same agent frequently This implies that we must try to get along with a few well-proved remedies instead of constantly trying new ones on our victims for no other reason than because somebody has recommended the drug Like a good painter we should have a certain 'palette containing the colors with which we are most familiar Remedies which are less well known we should save for those cases which fail to respond to the familiar ones. If we fail to stick to this rule we shall never gather sufficient experience This is a matter of great concern to the dermatologist because the number of recommended preparations is practically limitless.

It is very instructive to clarify the fateful role of coincidence by some examples. From the literature we know that in the early days of the injection treatment of varicose veins, when everybody feared embolism from this treatment one patient who for some fortuitous reason had *not* been injected died of embolism on his way home. When I did my first cisternal punctures, a patient whom for some reason I had *not* punctured on the same day developed a spinal lesion from a tuberculous spondylitis which had been diagnosed before Such coincidences can discredit a new method completely because it is difficult to conceive that there really is no relationship between such unusual events and the preceding operation

But on the other hand coincidence may also confer an entirely undeserved fame on an ineffective method of treatment One of my patients who had long been hospitalized because of a probable allergic eczema relapsed a few days after discharge as could be expected He was immediately hospitalized again but, considering the nature of the case I had every reason to fear another recurrence after his second discharge and 3 days later he actually appeared with an extensive papular eruption on his back At my wit s end I yielded to the fantastic

suggestion of a resident to try to cause an alteration in the reaction of the skin by injection of specially prepared blood from the patient. But since I did not have enough suitable glass tubes on hand the injection could not be given on the same day and the patient was told to return two days later When he returned the eczema had vanished and the patient stayed well during many months of observation.

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the fact that n the total number of cases is in the denominator and p the number of cures is in the nominator Therefore the larger the total number of cases the smaller the error On the other hand a larger number of cures *will increase the error caused by coincidence* The best control for the practical evaluation of the effect of a remedy is a large series of untreated or placebo-treated cases *a requirement* which is often difficult to fulfil.

There is another very important premise which is frequently overlooked in therapeutic observations namely *that the sample has not been slanted by an unconscious preselection of positive cases* Such a unilateral selection is liable to adulterate all those observations which are based on mere impressions, because the memory is much more likely to retain the positive cases than the negative This makes us appreciate the value of statistical counting which registers all cases with equal fidelity But even this does not avert all the danger because, even in collecting the cases for statistical evaluation an unconscious preselection may exert an influence For instance in a statistic based on ambulatory clinic patients the severe cases will be absent while a series based on hospitalized patients will lack the mild cases Often an unintended social preselection plays a pernicious part causing discrepancies between observations from private practice and those from out-patient clinics The possibilities of a biased selection of the sample have no limit and frequently they are difficult to recognize Therefore they must be viewed from all possible aspects and conclusions from statistical observations must be considered in the most careful manner

Finally the soundness of our observations is made difficult by the fact that the *conditions of the clinical experiment* lack both clarity and stability It is the great advantage of the laboratory experiment over clinical investigations that it permits us to increase our observations by repetition not only at will but also under the same conditions Since the conditions of the therapeutic observation are already difficult to assay we must take care not to add to the difficulties by *a complicated arrangement of the experiment* This means that we should use the unknown method of treatment which we want to appraise if possible alone and not simultaneously with other methods Simultaneous treatment with other effective methods invalidates every observation Polypragmasy as the simultaneous treatment with many methods is called may in emergencies be a duty of the physician As a rule however it is not in the best interest of the patient, and it deprives the physician of the possibility of gathering valid experience from his own therapeutic work This is particularly true of the dermatologist. In no other field of medicine is the custom of combining several and even many medications practiced as commonly as in the treatment of skin diseases The simultaneous use of vehicles and active agents alone is polypragmasy This however is unavoidable and therefore we must try to render it harmless by way of the 'bland starter treatment and if necessary by way

of the "bland intermediate treatment" the principle of which is the *successive* application of vehicle and medicament.

Another significant source of errors for the clinician is the *unreliability of the testimony of the witnesses* or in other words, the reports of the patients. We had emphasized that in our field the history plays an important part in the therapy. One should however never forget to evaluate the statements of patients with a grain of salt and a certain wariness. For instance, the statement of a patient that he does not tolerate salves is frequently prompted by the desire not to be treated with salves. Important as the affirmations of the patient are in guiding observations, it remains our obligation not to take them at face value but to check them carefully according to their importance.

Only he who has fully understood and mastered all these difficulties in acquiring experiences and only he who has made it a practice to take them into account will ever be a good therapist.

The beginning and the end of every dermatologic therapy and for that matter of every therapy is the *psychologic influence and guidance* of the patient. In many cases this part has to start by relieving the patient of any unfounded anxiety and worry. It is just the sufferers from skin diseases who consult their doctor so frequently because they constantly see their disease which keeps them in a state of concern. Thus patients with moles frequently consult the doctor not because of the mole itself but because they believe that it grows from month to month. If it is a *non-growing* mole, e.g. a large hairy nevus, or a mole the future behavior of which the physician cannot foresee with certainty it is always a good idea to make a good photograph, in order to demonstrate convincingly to the patient the stability of his condition at a later consultation. Of course, there are also skin diseases which the patient believes to be harmless but where quick interference is desirable or necessary such as precancerous keratoses and early stages of lupus vulgaris. In such conditions which are sometimes discovered only by chance, one has to explain the seriousness of the situation to the patient and insist on treatment.

Very often the skin patient himself as well as his family is *afraid of contagion*. Generally the layman has much more fear of non-contagious skin diseases such as eczema than of the most pernicious internal infections, such as pulmonary tuberculosis and influenza, because he *sees* more of the symptoms and is much impressed particularly by the secretion and scales. Therefore one frequently has to spend much time explaining to the patient that the dermatologist deals with much less contagious diseases than his colleagues in other fields. Even the tineas and impetigo contagiosa are really remarkably little contagious. Familial cases of trichophytosis, onychomycosis, and epidermophytosis are strikingly rare despite the well-known "bathing mycoses" and epidemics of barber's itch. Only the scalp mycoses in children are very contagious, representing the only example of a highly contagious familial dermatosis. On the other

hand there are mycoses of the skin which continuously scatter around masses of fungi-containing scales without even endangering the husband or wife more than other people This is the case in tinea versicolor which is a good example of a non-contagious infectious disease It is assumed that a relatively rare individual predisposition is a necessary factor in making the ubiquitous germ stick. Most of the contagious skin diseases require a particularly intimate contact for infection Trichophytosis of the bearded area is mostly caused by lack of shaving hygiene which practically equals an experimental inoculation. Impetigo contagiosa is mostly transmitted from the mother to the baby or vice versa and mutually only among *small* children because adults are much more careful about their contact with others. Scabies is mostly transmitted by bedding including hotel beds It most frequently affects only those members of the family who sleep in the same room, and it is relatively often transmitted by sexual intercourse so that it has jokingly been referred to as a fourth "venereal disease. And even the dreaded venereal diseases derive their name from the fact that they are *so little* contagious under ordinary circumstances that the infection mostly requires the most intimate bodily contact possible among adults. Extra-genital infections are rare exceptions under modern hygienic conditions. Contagion from the toilet seat usually is an alibi for the bad conscience of the patient. The doctor should have clear ideas about this generally favorable situation of skin diseases with regard to the danger of contagion so that he can effectively dispel the worries and phobias of his patients. Among the dermatoses there are of course many *chronic* diseases. Many patients think that chronic is synonymous with "incurable Therefore it is necessary to explain to the patients the medical meaning of chronic

The dermatologist also frequently encounters the apprehension that treatment would be of no avail because in most cases he is unable to give a satisfactory answer as to the *cause of the ailment* In such cases it is necessary to explain to the more intelligent patients that therapy is mainly an *empirical* science whose task it is simply to find out which methods of treatment and which drugs help and which *fail to do so* Thus there are diseases of completely unknown causes which however we are well able to influence as for instance lupus erythematosus with antimalarials and gold On the other hand we know the causative microörganism of some diseases very well but we do not know a medicament which could safely kill the germs in the body Examples of this latter situation are filariasis and many of the virus diseases.

The purpose of the psychologic guidance of the patient is not only to explain dangers and dispel unnecessary worries and unfounded pessimism It has a decisive significance for *the treatment itself* This does not mean that skin diseases can be healed by psychotherapy (again with some special exceptions, such as warts) but the patient s attitude toward our treatment is most essential for its success. In dermatology the co-operation of the patient is especially important

It is no exaggeration to say that the success of external and ambulatory treatment depends completely on the zeal and patience of the patient. By unauthorized interference and experimenting such as washing and treatment with other medications, the patient may mislead the judgment of the physician on the effectiveness of the individual salves and thus jeopardize the success of his treatment. Dermatologic treatment must start with building up confidence in the therapy and by explaining that the continuous prescribing of new salves is not an aimless experimenting but rather the well-tried principle of every methodical external treatment. In many dermatoses e.g acne psoriasis, chronic eczema lupus vulgaris epithelioma, it is essential for final success that the patient be not permitted to stay away when the symptoms have abated but that he be informed in time about the necessity of long posttreatment care and checkups. Of course, the interval between these checks should not be too short, in order to avoid unnecessary expense Patients who do not really care about the cure of their ailment, as happens with skin diseases quite frequently should not be accepted because they cannot be expected to put enough energy behind the execution of their external treatment to make it successful. For these reasons the results of treatment for non-paying patients such as relatives, friends, and members of compulsory health insurance plans, are, on the whole, much worse than in those who pay the normal fees. Understandably free advice is less well followed particularly if connected with trouble and inconvenience. This fact can even be observed on one's self I once had an intertriginous eczema which did not want to heal because I caught myself time and again skipping the application of the ointment which I had prescribed for myself. This hardly happens with patients who have to pay for their prescription, particularly if one keeps asking and prodding Thus the dermatologist must relentlessly do the utmost to enlist the full co-operation of the patient. In so doing, he will also succeed in clearing up even stubborn chronic skin diseases and in preventing recurrences or suppressing them at the start. These intelligent efforts will finally lead to a status which practically amounts to cure.

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